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AN EXPERIMENTAL STUDY OF THE EFFECTS OF DEPRESSED FRACTURES OF THE SKULL

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THIS enquiry into the effects of depressed fractures of the skull was prompted by the widespread opinion that such depressions invariably should be elevated. It often is stated that untreated depressions cause progressive brain damage and lead to pachymeningitis, adhesions, areas of cerebral softening, and brain cysts. To such changes post-traumatic epilepsy, insanity, and psychoses are attributed. Our experience causes us to question the correctness of such views.

HISTORICAL

Trephining for depressed fractures is an operation of antiquity (30). In the neolithic period, about 25,000 years ago, skulls were trephined. The operative mortality must have been exceedingly high. This operation was carried out also in ancient Egypt, as shown by a trephined skull described by Elliot Smith (30).

Of Greek surgery, in the period of 3800 to 500 B.C., there are no records. Without doubt, in the height of the Cretan civilization, 2000 to 1400 B.C., the Mycean civilization, 1500 to 1300 B.C., and the period of Homer, 1000 to 900 B.C., these operations must have been performed (4, 13).

The first important contribution to cranial surgery has been attributed to Hippocrates, 460 to 370 B.C. Hippocrates (18) did not favor operation in cases of depressed or comminuted fractures of the skull, but left the bone to suppurate gradually, thus discharging itself. He felt that fractures with depression were not particularly dangerous unless the membranes were ruptured. Severe contusions and simple linear fractures caused a great amount of cerebral vibration and would not allow the noxious material to come out of the brain. These he perforated to relieve the tightness of the brain and to procure evacuation of extravasated blood.

The Roman surgeon, Heliodorus (35), 75 B.C., expressed the idea that, in all cases of depressed fracture, the bones should be elevated, and Celsus, 30 B.C., agreed with him. Celsus (7), a prolific writer, approved of removing all spicules of bone immediately, elevating all depressed fractures, and smoothing down all protruding bone edges. He incised the scalp to inspect the condition of the outer table and, if necessary, the adjoining bone was trephined to elevate the depressed area. A meningophylox, an instrument designed for elevation, was used.

With some modifications, the ideas of Celsus held sway from 75 B.C. to 1811 A.D. Galen (35), 150 A.D., favored the use of gouges and the lenticular rather than the trephine. Oribasius (35), 325 to 403, and Paulus of Aegineta (35), 625 to 690, offered no new ideas but merely expressed the opinions of the earlier surgeons.

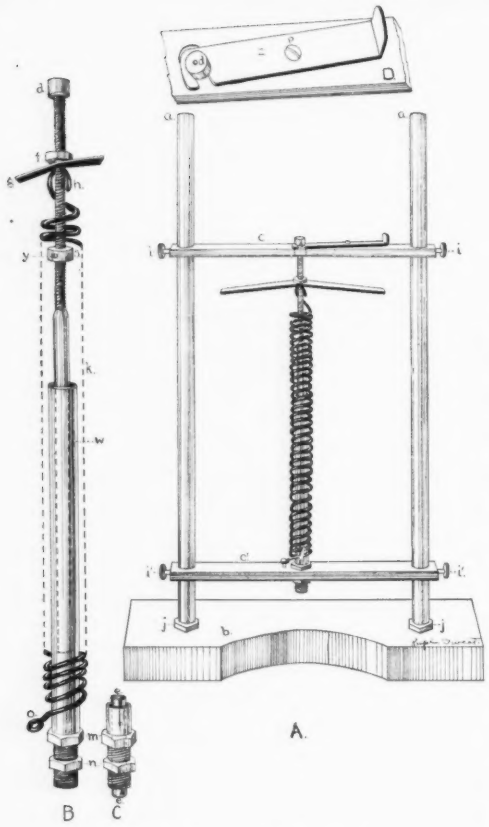

Fig. 1. Fracturing instrument.

The three Arabian physicians, Rhazes (17, 35), 860 to 932; Albucasis (17,35), 936 to 1013; and Avicenna (17,35) 980 to 1036, though favoring the elevation of depressed fractures, were skeptical as to the results obtained therefrom. They must have realized that the mortality was very high, for they said that only skilled surgeons should undertake this procedure.

The celebrated Jewish physicians, Avenzoar (35), 1162, and his pupil, Averrhoes (35), 1126 to 1198; also favored immediate elevation of depressed fractures.

Roger of Salerno (4), 1170, an important postclassical writer, believed it necessary to remove all the bone from areas of depression and to insert a fold of the finest linen with a feather, between the dura and the skull, so as

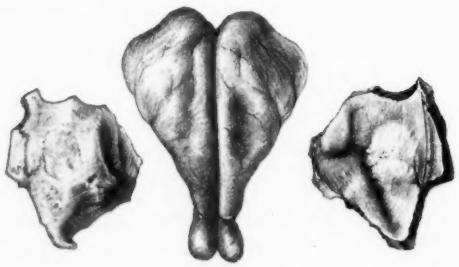

Fig. 2. Type A depression. There is a depression of both the inner and outer table, as well as of the underlying brain.

to clean the wound properly. In the opening of the fracture, a piece of clean linen or silk was so placed that the edges were beneath the bone. Marine sponges, carefully cleaned and dried, also were used to absorb any exuding matter. The dressings were changed twice a day in the winter and three times a day in summer. The external wound was covered with a piece of linen soaked in white of egg, lightly wrung out, over which a dressing of clean down was placed. The whole head then was bandaged with linen. It is very interesting to note the attention Roger gave to wound cleanliness.

Following the work of Roger of Salerno, Theodoric (4), 1205 to 1298, Walter of Agilon (4), 1250, and Mondeville (4), 1260 to 1320, also emphasized the necessity for the cleanliness of wounds. Theodoric, a pioneer in aseptic surgery, taught that pus was not necessary for the healing of wounds. Walter of Agilon believed that the skull should heal first and the skin last. Mondeville believed that all suppuration should be avoided.

The sixteenth, seventeenth, and eighteenth centuries brought forth many new and clearer theoretical considerations of brain damage. Ambrose Pare (34), 1509 to 1590; Jacobus Berengarius (4), 1728; Jean Louis Petit (36), 1674 to 1750; Percivall Pott (38), 1713 to 1788; John Hunter (33), 1728 to 1793; Jean Dominique Larrey (26), 1766 to 1842; all favored the elevation of fractures. Berengarius operated to prevent the advent of symptoms. Jean Louis Petit felt that the operation of trephining was not mortal and gave exit to

Fig. 3. Type B depression. There is a more marked depression of both the inner and outer tables and the underlying brain than in Figure 2. The depression of the inner table is perfectly rounded and smooth. There is no evidence of spiculation.

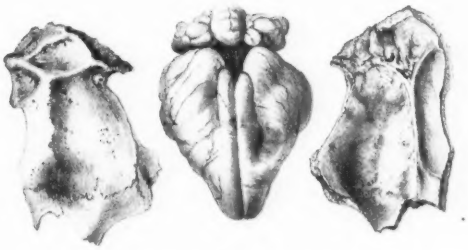

Fig. 4. Type B depression. Depression in the outer table has crossed the midline. There is a definite depression upon the surface of the brain. The inner table is again entirely smooth.

blood which had effused between the dura and bone. He thought that, even though all patients did not fall into this category, there was no way in which they could be differentiated, so all should be operated upon. Percivall Pott believed that the depressed bone was detrimental to the brain. In his opinion, if the depression was slight and there were no urgent symptoms and no injury to the membranes, elevation was sufficient; but, if the force had been great, cerebral damage was certain and, for this reason, the whole piece of bone was to be removed to allow free drainage and to relieve brain pressure. John Hunter believed that depressions of the inner table were usually greater than those of the outer table, and for this reason he advised elevation to prevent the possibility of any future harm. Larrey, the celebrated French military surgeon, considered immediate trephining indispensable.

Until the time of Abernethy (1), 1811, the surgeons were in fairly definite agreement. The entire nineteenth century was filled with divergent opinions, and it was not until the twentieth century that surgical opinion was unified again. Abernethy, the successor of Hunter in London, believed that many cases of depressed fracture were operated upon unnecessarily, and he cited 5 cases wherein depressed fractures existed without derangement of cerebral function. He believed that the brain was not such a delicate organ that the least degree of pressure was highly injurious. He was unable to say whether remote effects occurred, as he had not followed these patients for a long enough period of time. He could not conceive that pressure which caused no ill effects at the time of injury should produce injurious effects afterward, particularly when the brain had adapted itself to an altered size and shape. If symptoms of inflammation arose, he advised elevation to promote drainage.

Fig. 5. Type C depression. The depression of the inner and outer tables, as well as of the underlying brain, is more marked than the previous figures. Again the absence of spiculation is noted.

Fig. 6. Type D depression. The depression upon the outer table is well circumscribed. It may be noted that the inner table is spiculated and that this spicule has traumatized the brain.

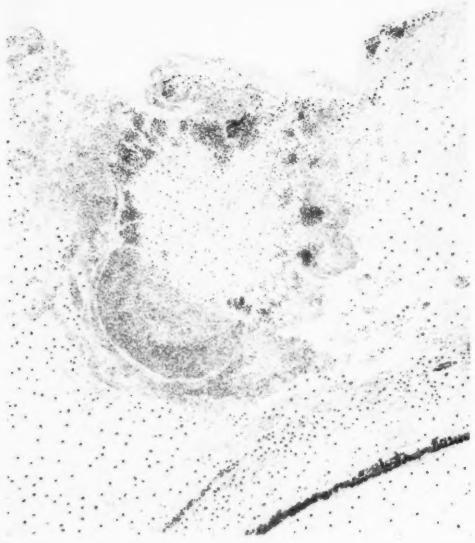

Fig. 7. Rabbit No. 56, 24 hours after fracture. Nissl toluidin blue. There is a hæmorrhagic area extending from the cortex to the corpus callosum. Polymorphonuclear leucocytes, mononuclear cells, and red blood cells are present. The depressed area may be noted on the surface of the brain.

The French school of this period favored non-operative treatment in simple depressed fractures, except when the symptoms of compression pointed to the depressed bone as their origin.

Thus two divergent views were held: one group favoring the elevation of depressed fractures and the other disagreeing.

Sir Astley Cooper (8), in 1839, was of the opinion that the cause of compression was the extravasation of blood, depression of bone and matter. He was the first to experiment upon the brains of animals. He applied digital pressure to the brain of a dog, noting that the greater the pressure, the slower the pulse, that, with slight pressure, pain and irritation were produced and that coma followed greater pressure. Upon release of this pressure, the dog entirely recovered. It was his belief that the mischief of depression was not always immediate. If an open wound was present he operated immediately, but, if there was no wound, the onset of symptoms was awaited.

Fig. 8. Rabbit No. 2, 4 days after fracture. Nissl toluidin blue. In the subcortical area new blood vessel formation has occurred, as well as glial proliferation. The insert shows the glial cells highly magnified.

It frequently happened that there was considerable depression of the external table with no damage to the internal table. Upon these patients he did not operate. Guthrie (15), 1842, elevated depressed fractures if they were over the parietal lobe, for he had seen patients whose symptoms cleared up immediately following this procedure. Thompson (9, 15) and Hennen (9, 15, 16) agreed with this view.

Laurie (18, p. 361), 1856, felt that all depressed fractures should be operated upon early to prevent serious consequences rather than await their development. In 1877,

Fig. 9. Rabbit No. 44, 6 months after fracture. Nissl toluidin blue. There is an area of necrosis in the subcortical area above and lateral to the corpus callosum. The ventricles may be noted as well as the depressed portion of the brain. The insert shows the large vacuolated glial cells.

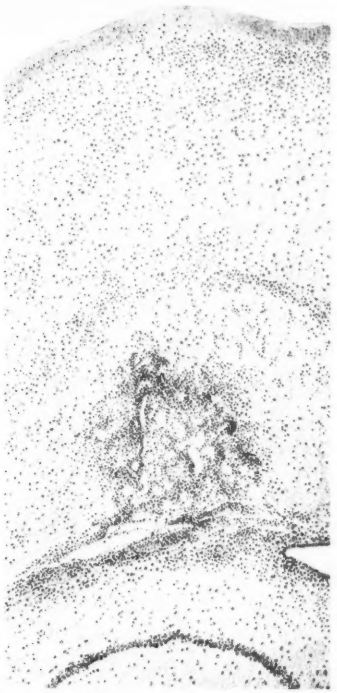

Fig. 10. Rabbit No. 28, 8 months after fracture. Nissl toluidin blue. The area of necrosis is superior to the corpus callosum. The tip of the lateral ventricle may be noted. Upon high power, gliosis and nerve cell degeneration are visible through the cortex and subcortical areas.

Robert Hudson (23) wrote his interesting paper in which he lamented the fact that, since the paper by Abernethy, trephining of depressed fractures had gone out of vogue. He fully realized the seriousness of the operation in London hospitals in which the mortality caused by pyæmia was very great. This he attributed to the septic atmosphere of the London hospitals. He gave the operative mortality as follows: Guy's Hospital, 51 of 67 operations; St. Thomas' Hospital, 3 operations and 3 deaths; St. George's Hospital, 13 deaths in 16 operations. In Redruth, Hudson said, trephining still was done, and the mortality was very low owing to the purity of the atmosphere which was daily swept by the Atlantic breezes.

In 1867, Lister (27, 28) delivered his celebrated lecture on asepsis before the British Medical Association in Dublin. The reduction of operative mortality and the fact that many sequelæ of non-elevated fractures were demonstrated led to the return of the operative trend. During the latter part of the nineteenth century, there was still variance of opinion and the subject was repeatedly discussed.

Holmes (14), in 1881, believed that simple depressed fractures should not be operated upon and, agreeing with him in this view, were Hewett (14, 19), 1881, Kinlock (14), 1882, Sands (42), 1883, and Nancrede (31), 1885. Hewett believed that, if the skin were broken, the fracture should be elevated. The severe symptoms were not caused by the depressed bone, but by the effusion of blood and the brain trauma. Sands hesitated to convert a simple into a compound fracture. Ashhurst (14) believed it better to leave a foreign body embedded in the brain than to add to existing irritation by injuring the brain by unsuccessful attempts at removal. Nancrede believed that elevation could not prevent the brain

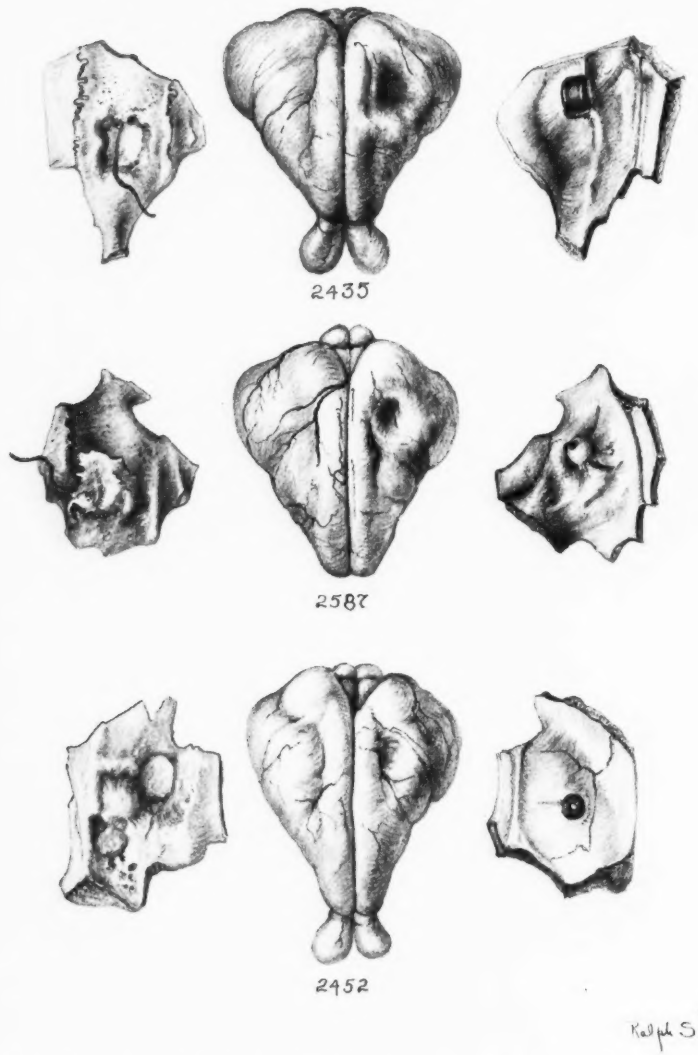

Fig. 11. Depressions of the brain. Beads inserted between the dura and skull resulted in marked cerebral depressions. The appearance of the outer table may be noted as well as the position of the bead in the inner table. These beads were held firmly in position by fibrous tissue growth.

damage caused by the depressed bone and felt that, for this reason, it was entirely unnecessary.

Levis (14), 1882, Gunn (14), 1882, Gross (14), 1882, Briggs (5), 1884, Roberts (40), 1884, Horsley (20, 21, 22, 28, 29), 1888, Keen (24), 1888, Macewen (29), 1888, Agnew (2), 1891, and von Bergmann (43), 1890, believed that fractures should be elevated but that certain limitations should be placed upon each case. Levis believed concussion could not be differentiated from compression and felt that the

depth of the depression should be used as the criterion for operation. Gunn thought that all depressed fractures, whether simple or compound, should be elevated if it seemed probable that the inner table was depressed. Gross said that, if the depressions were moderate, they should be left alone; however, if there were symptoms indicative of depressed inner table, operation should be performed. Briggs agreed with Gross and Gunn. Horsley felt that all cases of depressed fracture could not be benefited by elevation after traumatic epilepsy had occurred, but advised that, in all early cases, the depressed fracture should be operated upon to prevent epilepsy. In this opinion, Macewen, Agnew, and von Bergmann concurred. It now may be noted that the elevation of depressed fractures has taken on a definite purpose: that of preventing late results. All have agreed that, after these late symptoms have arisen, operative interference is of little value.

The twentieth century writers are unanimous in the foregoing opinion but their ideas as to the definite cause of these post-traumatic symptoms are still extremely hazy. Charles Phelps (37), in 1900, Crisp English (12), 1904, Pearce Bailey (3), 1906, E. Krause (25), 1909, Keen (10), 1915, B. Rawling (39), 1919, Charles Kahlke (32), 1920, Butler (6), 1921, Eagleton (11), 1921, and Ernest Sachs (41), 1922, are among the writers upon this subject.

With fractures which have lacerated, torn, or penetrated the dura and cortex, one expects to find varying degrees of change such as thickening of the meninges, adhesions, scars, and degeneration of the cortex, and such has been our experience. If the dura is opened, some evidence of injury to the cortex may be apparent even following simple depressions. However, such changes are not always present; on several occasions we have found a normal dura beneath such a depression. Opening the dura revealed no discernible pathological change. The dura was not thickened, the leptomeninges were of normal appearance and not adherent, and the color and circulatory condition of the brain cortex seemed normal. Such negative findings were noted not only in recent simple depressions but also in some of those of several years' standing.

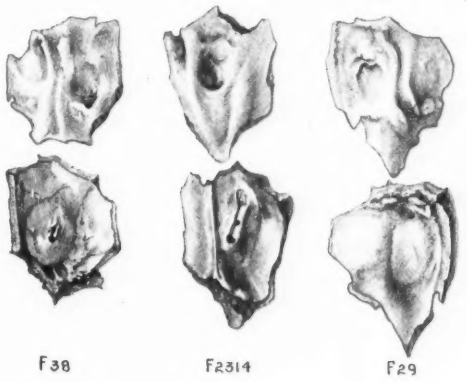

Fig. 12. This is a series of skulls showing the relation of the damage of the outer table to the inner table. F-38 shows a well rounded depression of the outer table with fragmentation of the inner table. This may also be noted in F-2314. F-29 demonstrates a circumscribed depression of the outer table with a well rounded depression of the inner table.

Such experiences led us to question the wisdom of our operative intervention and prompted an enquiry into the relation of depression to changes in the brain. Are pathological changes, when found, the direct effect of the trauma or are they caused by the continued depression and in what way may these changes be modified by removal of the depression?

In an endeavor to establish a solution of this problem, the following experimental work was outlined: a series of depressed fractures were to be produced in rabbits and, at varying intervals of time following fracture, the brains were to be studied. As a control, depressed areas of dura and brain were produced in other rabbits by the insertion of glass beads between the dura and the skull.

OUTLINE OF EXPERIMENTAL WORK

The conduct of this experiment necessitated:

1. Procuring a device which would produce identical depressed fractures in a series of animals.

2. (a) Pathological studies of the brains of animals at varying periods of time after the receipt of a depressed fracture. (b) Comparison of such changes with those produced by a depression not associated with a blow.

TABLE I.—TYPE A

Animal No.	Weight Kilos	Sacrificed at	Pathological changes	
			Gross	Microscopic
74	2.1	3 hours	Very slight hæmorrhage beneath the pia. Slight flattening over area of depression	Evidence of hæmorrhage in the meninges, a slight hæmorrhage over the cortex with a slight degree of œdema.
73	1.9	6 days	Slight flattening over area of depression	No pathological change.
8	3.6	6 days	Dura adherent to bone, but appeared normal. In the region of fracture was a slight discolored area with a slight depression. Veins were slightly dilated on the injured side	No pathological change.
6	3.2	7 days	Slight flattening over area of depression	No pathological change.
72	2.4	7 days	Slight flattening over area of depressed bone	No pathological change.
14	1.7	8 days	Vessels on the injured area appeared slightly dilated. Slight flattening over the area of depressed bone	No pathological change.
20	2.3	20 days	Slight area of depression	No pathological change.
61	2.0	21 days	Slight flattening over area of depressed bone	No pathological change.
15	2.6	26 days	Vessels on the injured side slightly dilated. Slight flattening over the area of depressed bone	No pathological change.
41	4.5	2 months	Slight brownish tinge over area of fracture with the vessels on the injured side slightly contracted. Slight flattening over area of depressed bone	No pathological change.
52	3.5	2 months	Slight flattening over area of depressed bone	No pathological change.
16	2.3	6½ months	Slight flattening over area of depressed bone	No pathological change.
32	2.1	8 months	Slight flattening over area of depressed bone	No pathological change.
39	3.5	9 months	Slight flattening over area of depressed bone	No pathological change.
17	2.5	12 months	Slight flattening over area of depressed bone	No pathological change.
55	2.7	12 months	Slight flattening over area of depressed bone	No pathological change.

3. (a) Study of the brains in which a localized depression was produced by the extradural insertion of a small foreign body (bead). (b) Comparative study of a group in which such depressions had been produced and later relieved after varying periods.

APPARATUS

In order to produce experimentally a series of similar, simple, depressed fractures in animals, it was necessary to devise an instrument by which the same traumatic force and the same depth of cranial depression could be produced on each occasion. Figure 1 shows the instrument devised for this purpose. This instrument consists of an iron plunger, *e*, with a rounded tip, which travels in the cylinder *w*. The cylinder, *w*, is permanently attached to the cross beam, *c-1*, by the nuts, *n* and *m*. The force is regulated by the spring, *k*, and is directly proportional to the stretch of the spring. This stretch may be varied and is controlled by the distance between the two cross beams, *c* and *c-1*.

By trial the required force was determined. This was 10.5 inches of spring stretch. As the traumatic force had little to do with the depth of depression, the length of protrusion of the plunger, *e*, next had to be ascertained. This was five-eighths of an inch. Set screws, *y* and *s*, hold the plunger at this position.

Rabbits were used as experimental animals. The head was shaved and the rabbit was anæsthetized. The head was placed upon the

base, *b*, with the neck fitting into the circular indenture. The base of the cylinder, *2*, was placed directly in contact with the rabbit's skull to one side of the midline and three-quarters of an inch from the occipital protuberance. The cross beam, *c-1*, was firmly screwed at the level thus obtained. The cross beam, *c*, was then raised so that the distance between *c-1* and *c* was 10.5 inches. The plunger was locked into position by the trigger, *z*. With the release of this trigger, the plunger was forced downwa.d, through the cylinder, *w*, with a traumatic force equal to 10.5 inches of spring stretch and striking to a depth of five-eighths of an inch.

EXPERIMENTAL PRODUCTION OF DEPRESSED FRACTURES FOR PATHOLOGICAL STUDIES

Early in the course of these experiments it was found that the skulls of different rabbits varied in strength, and for this reason the depth of depression of the inner table varied. It was then necessary to classify these rabbits into various groups depending upon the depth of depression. This classification was determined entirely by the autopsy findings and was as follows:

Type A—very slight rounded depression without dural injury.

Type B—marked depression without dural injury.

Type C—greater depression than type B without dural injury.

Type D—severe depressions with comminution; dural and brain laceration.

Following fracture, all the animals except a few in the type D class rapidly recovered. There was no evidence of any clinical symptoms and all the animals appeared entirely normal. These animals were then sacrificed at varying intervals, the groups being tentatively determined by the palpation of the depressed area. The brains were then examined, both grossly and microscopically, for evidence of pathological change.

Type A (Fig. 2) demonstrates the slight depression in the inner and outer tables of the skull and upon the surface of the brain. There was no evidence of spiculation.

Table I represents a protocol of the 16 rabbits placed in this series. Upon study of this table, it will be noted that these animals were sacrificed at periods from 3 hours to 12 months after the injury. In all of these cases there was a slight definite depression upon the surface of the brain. Only one animal presented any evidence of intracranial damage and this consisted of a slight surface hæmorrhage. This animal was sacrified at 3 hours. There was no evidence of late pathological change in any.

The illustrations of Types B and C (Figs. 3, 4, and 5) show the increased depth of fracture and brain depression. It may be noted that the depression in the brain is much greater than in type A.

Table II represents a protocol of 31 rabbits which comprise this series. A study of Table II shows that these animals were sacrificed at periods of time from 30 minutes to 9 months after fracture. In each there was a definite deep depression upon the surface of the brain and there was no spiculation of the inner table. Gross examination of these brains did not reveal any definite pathological changes, but microscopic examination presented some very interesting facts. The pathological changes shown varied with the time since injury.

For a comparison of the brain damage and the evidences of repair, three groups may be considered: Group I, acute traumatic—one-half hour to 30 days; Group II, interval period—30 days to 90 days; Group III, late pathological changes—90 days to 9 months.

Group I. In this group there were 15 rabbits, sacrificed from one-half hour to 30 days after fracture. All the evidence of acute trauma with the resulting repair was present. The meninges showed definite hæmorrhage, with the presence of red blood cells, leucocytes, eosinophiles, and small mononuclear cells. Macrophages were present also, as well as were dilated, engorged, and ruptured blood vessels.

Within the brain substance were varying degrees of hæmorrhage, from slight subpial hæmorrhage to a large area of hæmorrhage which extended from cortex to ventricle. The cellular infiltration consisted of red cells, polymorphonuclear leucocytes, mononuclear cells, some of them vacuolated and others containing brownish pigment. Many fat laden

TABLE II.—TYPES B AND C

Animal No.	Weight Kilos	Sacrificed at	Pathological changes	
			Gross	Microscopic
60	1.5	½ hour	Vessels over the area of depression were dilated, the dura was intact. Definite flattening over area of depression	Marked extravasation of blood beneath the pia. The small vessels were ruptured. This existed only over the area of depression and was well localized. There was evidence of œdema.
63	2.5	½ hour	Same as rabbit No. 60	Same as rabbit No. 60.
56	2.0	24 hours	Same as rabbit No. 60	The hæmorrhage was more severe than in rabbit No. 60; it extended deeply into the brain substance as far as the ventricular wall. The œdema was more marked.
26	2.0	24 hours	Same as rabbit No. 60	Marked congestion of the vessels with some chromatin degeneration. The cellular infiltration consisted of practically all red cells, very few leucocytes, and some mononuclear cells.
2	2.7	4 days	Same as rabbit No. 60	Subpial hæmorrhage was present. There was also extravasation of blood to the ventricular wall. One vessel at the marginal area showed perivascular infiltration of small round cells. Œdema was marked and there were many large mononuclear cells present; some of them were vacuolated and some contained brownish pigment. The capillaries were very distinct in the center, but at the border line they were suggestive of endothelial proliferation. Many fat laden cells were present in and about the hæmorrhagic area. The nerve cells were in a stage of chromatolysis. The glial cells were swollen and were in various stages of degeneration. There was a slight tendency to glial reaction. Particularly in one part there was a definite zone of gliosis consisting almost entirely of glial nuclei. Macrophages were present in large numbers. In the meninges were also many small mononuclear cells containing pigment. The macrophages were present here also but not in so great numbers as in the brain.
7	3.4	7 days	Over the area of depression upon the surface of the brain was a discolored region	In this brain was noted a lessening of the number of large macrophages and a great decrease in the red cells but with an increase in the number of mononuclear cells. A glial reaction was still present but this was much less than in rabbits sacrificed after four days.
3	2.9	7 days	No evidence of injury on the surface of the brain	Hæmorrhage within the meninges and gitterzellen were found in rows along the vessels. There were a number of eosinophilic cells in the region of the hæmorrhage.
62	3.0	7 days	Same as rabbit No. 7	Same as rabbit No. 7.
12	3.0	9 days	Area of depression well localized	Same as rabbit No. 7.
33	2.5	12 days	Area of depression well defined, slight discoloration on surface of depression	No pathological change.
48	3.5	13 days	Area of depression well defined	Pathological changes very similar to those of rabbit No. 7, but fewer signs of active damage. Eosinophiles more abundant.
13	3.5	20 days	Area of depression well defined	More marked absorption and regeneration of tissue.
46	3.4	24 days	Area of depression well defined	No pathological change.
31	1.5	28 days	Area of depression well defined	No pathological change.
30	2.3	30 days	Area of depression well defined	Showed a slight persistence of old hæmorrhage. There seemed to be gradual absorption.
4	3.6	34 days	Area of depression well defined. Veins slightly dilated on injured side	No pathological change.
34	2.5	34 days	Area of depression well defined	No pathological change.
69	2.5	35 days	Area of depression well defined	No pathological change.
21	2.3	28 days	Area of depression well defined	No pathological change.
40	3.5	69 days	Area of depression well defined. Depressed area somewhat whiter in color than remaining brain	No pathological change.
42	3.5	77 days	Area of depression well defined. Slight graying of cortex over injured area	No pathological change.
45	4.0	90 days	Area of depression well defined	Deep in the brain substance, above the corpus callosum, was a well marked area of necrosis with œdema and vacuolated cells. Some of the cells were degenerated, chromatolysis was present in the nerve cells, and very few glial cells were visible. There were fat containing macrophages.

TABLE II.—Continued

Animal No.	Weight Kilos	Sacrificed at	Pathological changes	
			Gross	Microscopic
38	5.4	90 days	Area of depression well defined	Same as rabbit No. 45.
29	2.1	95 days	Area of depression well defined	Same as rabbit No. 45.
43	4.3	97 days	Area of depression well defined	Same as rabbit No. 45.
37	4.5	5 months	Area of depression well defined	Same as rabbit No. 45.
36	4.5	6 months	Area of depression well defined	There was an area of necrosis against the corpus callosum with many gitterzellen and glial cells. Between the cells there was a granular amorphous substance especially in the outer part of the area. Surrounding the necrotic region there was a glial proliferation and a few scattered gitterzellen. Large reacting glial cells were present about the area of necrosis. Between the cortex and this area of necrosis, there was an increase of irregularly distributed small glial cells. A few of the nerve cells showed chromatolysis.
50	3.0	6 months	Area of depression well defined	Same as rabbit No. 36.
44	3.5	6 months	Area of depression well defined	Same as rabbit No. 36.
28	2.2	8 months	Area of depression well defined	Area of necrosis was extending nearer to the cortex than in the previous rabbits.
49	3.5	9 months	Area of depression well defined	Area of necrosis extending nearer the cortex.

TABLE III.—PRESSURE DEPRESSED FRACTURES

Animal No.	Weight Kilos	Sacrificed at	Pathological changes	
			Gross	Microscopic
23	2.3	24 hours	Area of depression defined	No evidence of pathological change.
64	2.7	8 days	Area of depression defined	No evidence of pathological change.
27	2.1	10 days	Area of depression defined	No evidence of pathological change.
19	2.3	2 months	Area of depression defined	No evidence of pathological change.
59	2.5	4 months	Area of depression defined	No evidence of pathological change.
58	2.5	12 months	Area of depression defined	No evidence of pathological change.

cells were present in and about the hæmorrhagic area. Macrophages were present in great abundance. The vessels showed a perivascular infiltration of round cells, and there were gitterzellen in rows along them. At the border line between the normal brain structure and the injured area, the capillaries were swollen suggesting endothelial proliferation. The nerve cells were very often in the stage of chromatolysis. The glial cells were swollen and were in various stages of degeneration. In other areas there was a definite gliosis. Œdema was marked throughout (Figs. 7, 8).

Group II. This series comprised 6 rabbits, sacrificed at intervals from 34 to 90 days. No gross pathological changes were present. The only microscopic evidences of pathological change were the presence of occasional pigmented cells, the remains of early hæmorrhage. It can be noted that within this period of time, the early evidences of trauma disappeared.

Group III. This group of animals consisted of 10 rabbits sacrificed at intervals varying from 90 days to 9 months. Again there were no gross evidences of pathological change. Microscopically, however, there were some striking changes. The cortex and meninges were normal. In the subcortical areas, near the corpus callosum, was definite evidence of brain necrosis. Surrounding this necrotic area there were glial proliferation and many gitterzellen. Between the cells was a granular amorphous substance, particularly in the outer areas of this necrotic region. A great number of large reacting glial cells were scattered about. Between the cortex and this

TABLE IV.—EXPERIMENTAL PRODUCTION OF DEPRESSED BRAIN WITHOUT FRACTURE

Animal No.	Weight Kilos	Sacrificed at	Size of bead inserted, mm.	Pathological changes	
				Gross	Microscopic
15-b	3.6	2 days	2	Area of depression well defined	No evidence of pathological change.
19-b	3.5	8 days	2	Area of depression well defined	No evidence of pathological change.
28-b	2.4	18 days	3½	Area of depression well defined	No evidence of pathological change.
10-b	3.2	19 days	2	Area of depression well defined	No evidence of pathological change.
5-b	3.2	20 days	5	Area of depression well defined	No evidence of pathological change.
11-b	3.1	21 days	2	Area of depression well defined	No evidence of pathological change.
1-b	3.2	30 days	5	Area of depression well defined	No evidence of pathological change.
25-b	3.2	2 months	2	Area of depression well defined	No evidence of pathological change.
13-b	2.7	3 months	2	Area of depression well defined	No evidence of pathological change.
7-b	3.4	3½ months	5	Area of depression well defined	No evidence of pathological change.
20-b	2.8	14 weeks	2	Area of depression well defined	No evidence of pathological change.
12-b	3.2	5 months	2	Area of depression well defined	No evidence of pathological change.
24-b	2.6	5½ months	2	Area of depression well defined	No evidence of pathological change.
9-b	4.1	6 months	2	Area of depression well defined	No evidence of pathological change.
2-b	3.2	9 months	2	Area of depression well defined	No evidence of pathological change.
21-b	3.8	10 months	2	Area of depression well defined	No evidence of pathological change.

TABLE V.—OPERATIVE REMOVAL OF BEADS

Animal No.	Weight, Kilos	Period bead removed	Postoperative sacrifice, months	Animal No.	Weight, Kilos	Period bead removed	Postoperative sacrifice, months
16-b	3.5	1 day	5	6-b	3.2	12 weeks	6
18-b	3.6	4 days	5	14-b	3.4	13 weeks	6
22-b	3.7	7 days	5	23-b	2.8	14 weeks	5
4-b	3.2	20 days	5	8-b	3.0	5 months	4
26-b	2.3	2 months	4				

necrotic area there was a slight increase of glial cells and some of the nerve cells showed chromatolysis. In the rabbits sacrificed at a later period, it appeared as if this necrotic area more nearly approached the cortex. It is very important to note that the gross appearance of the cortex or meninges in no way foretold the conditions existing in the subcortical areas (Figs. 9, 10).

Type D: In the course of our experimental work, it so happened that, in 12 rabbits, fractures of such severity were produced as to lacerate the brain and dura. These were not utilized for this problem, but we mention them in passing. In Figure 6 will be noted a well rounded depression of the outer table, with spiculation of the inner table. From the general appearance of this outer table, it would be impossible to ascertain the existence of this spiculation. Upon the brain was noted a well marked area of necrosis in addition to the area of depression.

EXPERIMENTAL PRODUCTION OF DEPRESSED FRACTURES

By pressure. This group consisted of 6 rabbits in which depressions of the brain were caused by pressure applied to the plunger which then slowly crushed the thin bone of the rabbit's skull. In this manner, a depressed fracture was produced without striking a blow. Table III presents a protocol of the 6 rabbits of this series.

Upon study of Table III, it will be noted that these animals were sacrificed at a period of 24 hours to 12 months. In all of these animals there was a definite depression in the bone and the underlying brain without evidence of dural or brain laceration. The microscopic examination was entirely negative

as to acute, traumatic pathological change and the late deep changes observed in Types B and C were absent also. From this observation, it is evident that localized pressure against the brain and meninges from depressed bone did not cause discernible morphological changes.

In this experimental work, when a simple depressed fracture was produced, the depression of the inner table tended to be less marked than that of the outer table. However, it was found that spiculation and cracking of the inner table may readily exist without evidence of such a condition of the outer table (Fig. 12).

EXPERIMENTAL PRODUCTION OF DEPRESSED BRAIN WITHOUT FRACTURE

The foregoing experiments have shown that depressed fractures caused by striking a blow produced both early and late pathological changes. Depressed fractures produced by pressure did not produce such changes.

In addition it was decided to produce varying depressions in the brain by the surgical introduction of small glass beads between the dura and the skull.

Under ether anæsthesia and with aseptic precautions, a small hole was trephined in the rabbit's skull. Glass beads were inserted through this opening and moved forward extradurally so as to remain *in situ*. Three sizes of beads were utilized, namely 2 millimeters, 3.5 millimeters, and 5 millimeters (Fig. 10). Each of these beads produced a greater depression upon the surface of the brain than our deepest "C" fracture. Table IV represents a protocol of the 16 rabbits comprising this series.

In the study of Table IV it will be noted that these rabbits were sacrificed at from 2 days to 10 months. After the placing of the beads, there was a deep depression upon the surface of the brain. Gross examination did not reveal any pathological changes. Microscopic examination revealed no changes other than some condensation of the brain tissue beneath the corresponding pit. There was no difference in the microscopic examination of the animal sacrificed at 2 days and that of the animals killed later.

Operative removal of beads. It was originally thought that possibly depressions caused by beads might produce pathological change and for this reason, a control series of 9 operative animals was used. Table V represents a protocol of the rabbits placed in this series. Upon study of this table, it will be noted that these animals were sacrificed from 4 to 6 months after the removal of the beads. These beads were removed at varying intervals of 1 day to 5 months. The brains, upon examination, showed the absence of pitting which the beads had created previously. Microscopic examination gave no evidence of condensation of the brain tissue in the position where the bead had been embedded.

CONCLUSIONS

Studies of the effects of simple, non-penetrating depressed fractures of the skull in rabbits have shown that:

1. The changes in the brain are caused by the force producing the injury rather than the depression of the bone.
2. The pathological changes in the brain appear more marked in the early and in the late stages than during the intermediate period and are chiefly subcortical.
3. Depressions of moderate size cause no pathological changes in the underlying meninges and brain.
4. Following the production of depressed areas by slow localized compression or by the introduction of extradural foreign bodies, pachymeningitis, leptomeningitis, adhesions, softening, or cysts were not produced.

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AN ANATOMICAL STUDY OF SUBDURAL HÆMORRHAGE ASSOCIATED WITH TENTORIAL SPLITTING IN THE NEWBORN

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INTRACRANIAL hæmorrhage, associated with a tearing (or more correctly a splitting) of the tentorium cerebelli, in the newborn has anatomically acquired an increasing importance in recent years, while clinically and surgically it has received as yet very little consideration. It is the purpose of this communication to contribute an etiological and anatomical analysis of a series of 32 cases of subdural hæmorrhage associated with tentorial splitting, first, as regards the lesions, and second, as regards their clinical significance.

ANATOMICAL INTRODUCTION

In order to appreciate the possible source of the subdural hæmorrhage and the formation of pathological lesions in the tentorium cerebelli, it is desirable to have a clear mental picture of the normal anatomy of the dura mater septa and its veins and sinuses.

The dura mater consists of two distinct layers. An outer, which is very vascular and serves as the bony periosteum, and an inner, which is but slightly vascular and may be considered the dura proper. It is the inner layer only which is prolonged inward to form the falx cerebri, the falx and tentorium cerebelli. These septa take origin along the venous sinuses which are themselves formed by clefts in the inner layer of the dura. They have a point of common union at the confluens sinuum (torcular hirophili). As the name indicates it is the meeting-place of the superior sagittal, straight, occipital, and two transverse sinuses. It is excellently protected posteriorly by the thick internal occipital protuberance and on all other aspects by thick radiating fibers that form the dural septa.

On examining these septa one is at once impressed by the fact that they are not of equal thickness, but in definite places condense to thickened bands. Holland is convinced that these "strengthening bands" have a definite protective function which is of highest importance during the stress of labor. The tentorium cerebelli contains more of these strengthening bands than any other reflection of the dura mater, and it is subjected to extreme strain during the stress of labor. There are (1) a curved band running obliquely downward and forward forming the anterior free boundary of the tentorium cerebelli, which is formed by the splitting of the fibers of the falx cerebri and is attached to the anterior clinoid process on either side; (2) a straight band running downward and backward at the junction of the tentorium cerebelli and the falx cerebri, a thickening which forms the floor of the straight sinus; (3) a curved band running slightly outward from the anterior boundary of the confluens sinuum and then horizontally forward to join the anterior free margin near its attachment to the anterior clinoid process (Fig. 5). These fibers are necessarily deep to the oblique fibers and do not form a boundary to the tentorium cerebelli. These three bands outline a triangular space which is, in the great majority of cases, the site of tentorial splitting (Fig. 4). From the point of origin of the anterior free margin superficial fibers radiate out like a fan to their insertion along the superior margin of the petrous temporal side and extend back to form the anterior wall of the confluens sinuum. Similarly, from the point of origin of the horizontal band, deep fibers radiate out less abundantly from the confluens sinuum. They cross the superficial fibers and finally join them at the free margin near the anterior clinoid process. The tentorium cerebelli is thus formed by two radiating systems of fibers: (1) a superficial, extensive, and predominatingly vertical system, referred to as the superficial tentorial blade; and (2) a deep, rather sparse, and predominately horizontal system, referred to as the deep blade of the tentorium cerebelli.

METHOD

The method employed for the examination of the intracranial contents in this series of cases is based on that originally described by Beneke (2) in 1910. We employed the slight modification which appeared in a later paper by Beneke and Zausch (3). This method permitted a more careful examination of the great cerebral vein and its tributaries. The cerebral hemispheres were ablated, care being used to preserve the posterior part of the third ventricle and the corpora quadrigemina. The falx cerebri and longitudinal sinus were preserved in every instance by making incisions 1.5 centimeters parallel to either side of the sagittal suture line. These incisions were carried forward through the frontal bone to meet the anterior extremity of the lateral incisions just above the orbital plates. The infratentorial basilar structures were examined by removing triangular windows from the occipital bone just below the lateral sinuses on either side of the midline.

This technique has the advantage of permitting a good exposure of the supratentorial fossæ and of all the reflected dural septa, with a minimum of manipulation. It, therefore, permits a careful examination of the most common sites of subdural and leptomeningeal hæmorrhage, as well as of the dural septa, and it greatly reduces the liability of postmortem tears (artifacts). The marked increase in the percentage of intracranial lesions in the newborn which have been recorded in recent years is probably more apparent than real. It is due partly to the general adoption of this technique.

The anatomical evidence for prematurity must be carefully considered because this represents by far the most important predisposing factor to both intracranial hæmorrhage and tentorial splits. Absolute evidence of prematurity is very difficult to establish. There can be no absolute certainty that the age of the individual fetus has been correctly estimated. None of the three methods usually employed is entirely satisfactory. In order of their importance Cruickshank and Miller enumerate these methods as follows: (1) measurement of body length and weight; (2) measurement and enumeration of centers of ossification; (3) the menstrual history of the mother. The measurement of body length and body weight is obviously limited in value by differences in the state of nutrition in fetuses of the same age. This is readily shown by Browne's length-weight ratio (obtained by dividing the weight by the length.) Hess has tabulated and compared records of fetal lengths and weights which further demonstrate the unreliability of records of fetal weights as compared with records of fetal lengths as an indication of prematurity. He finds the average fetal length at the end of the tenth lunar month to be 48.4 centimeters. We have, therefore, accepted a length of 48.0 centimeters and over as the most reliable anatomical evidence of maturity in the present study. We have compared this anatomical evidence with the clinical evidence as indicated by the menstrual history of the mother. In our series of 32 cases of tentorial splitting, we find that 13 infants were premature as indicated by the mother's history, and that the same number were premature as indicated by the length of less than 48.0 centimeters. In 2 cases, however (7 and 10), the clinically premature infants measured 48.0 centimeters or more, and in 2 other cases (28 and 31) the clinically full term infants measured slightly less than 48.0 centimeters. We have, therefore, a similar small margin of error, whether we accept the clinical or the anatomical evidence for prematurity.

PATHOLOGICAL FINDINGS

Subdural hæmorrhage. In this series of cases it is of significance to note that the intracranial hæmorrhage was almost completely confined to the subdural space. A classification of subdural hæmorrhage is unavoidably an arbitrary one because of the difficulty of accurately judging the diffuseness and quantity of the blood. The hæmorrhages were grouped as small, moderate, and large. This purely arbitrary standard was established after a comparative study of all the cases.

In regard to the amount and position of subdural hæmorrhage four significant points relative to its source were noted: (1) Slight or moderate amounts of blood were usually confined to the posterior (occipital) and middle

Fig. 1. Transverse section through the tentorium cerebelli showing evidence of stretching. Note the separation of the superficial vertical fibers and thinness of the deep horizontal fibers. ×34.

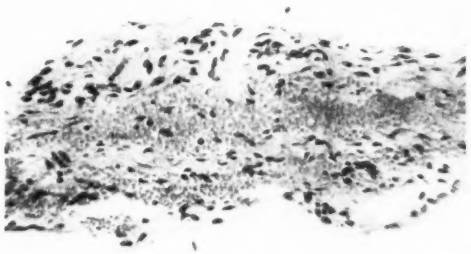

Fig. 2. Tentorium cerebelli in an 8 months premature stillborn infant. Note the uniformly immature fibrous connective tissue cells and the extravasated red blood cells between the tentorial layers. ×110.

(temporal) fossæ, while large amounts of blood usually include also the anterior fossa. (2) In nearly every instance blood was found in the subdural space either surrounding the great cerebral vein or its tributaries close to its junction with the straight sinus. In no instance, however, were we able to demonstrate an actual rupture of the great cerebral vein. (3) In every instance but one the subdural hæmorrhage was very largely confined to the supratentorial fossæ. In this instance (Case 29) there was, in addition to the supratentorial hæmorrhage, extensive clot surrounding the base of the brain. This was a case of podalic version with extraction in which there was evidence of stretching of the ligaments between the base of the skull and the atlas and axis. The anatomical picture indicated that torn meningeal basilar vessels were the source of the subdural hæmorrhage in this instance. (4) There was no definite relation between the degree of tentorial damage and the amount of subdural hæmorrhage. (See analysis of cases.)

In the light of these observations what is the usual source of the subdural hæmorrhage in the newborn? There are three sources to be considered in relation to tentorial lesions: (1) small intratentorial vessels; (2) emissary veins; (3) tributaries of the great cerebral vein.

In an attempt to determine the venous supply of the tentorium cerebelli and other dural septa, an injection of the great sinuses and their tributaries was made in a series of 10 cases. The injecting fluid used was india ink in a suspension of 15 per cent glycerine. After being washed for 20 minutes with normal saline, the longitudinal sinuses were injected through the anterior fontanelle. The fluid was allowed to escape through incisions in the jugular veins. These preparations were then hardened in 6 per cent formalin after which the dural septa were exposed in the usual way. The following observations are the result of a careful gross examination of these specimens:

1. There is, in many cases, a small intratentorial vein running more or less parallel to and just below the deep horizontal band. It appears to join the posterior end of the straight sinus (Fig. 5).

2. Relative avascularity of the tentorium in the full term infant as compared to the definitely premature fetus.

3. Complete absence of emissary veins in the triangular space.

4. Several tributaries joining the great cerebral vein as it approaches the anterior end of the straight sinus.

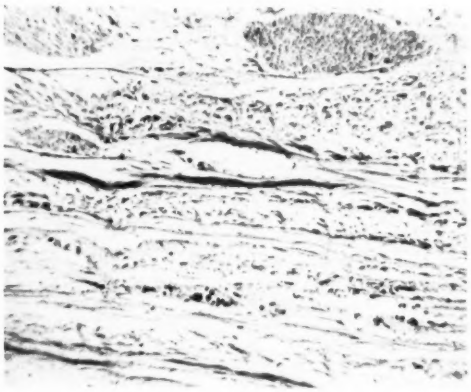

Fig. 3. Tentorium cerebelli in a full term stillborn infant. Note the maturity of the fibrous connective tissue cells as compared with the picture in Figure 2. ×100.

Fig. 4. Sagittal section through infant's head. *A* indicates the inferior surface of the triangular space of the tentorium cerebelli. This is the most common position of tentorial splitting.

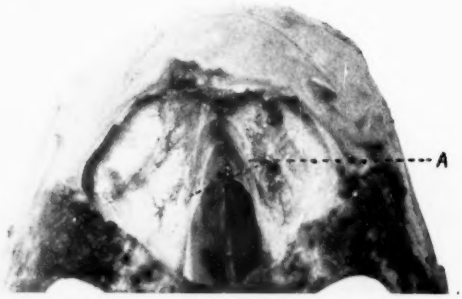

Fig. 5. Tentorium cerebelli in full term infant viewed from below. Note the position of the injected intratentorial veins. *A* indicates deep horizontal bands.

From these observations we conclude that extensive subdural hæmorrhage from ruptured intratentorial veins (in the presence of a split tentorium) is only probable under two conditions: definite prematurity and rupture of the intratentorial tributary of the straight sinus below the deep horizontal band. This vessel, lying below the triangular space, can be ruptured only in very extensive or perforating tentorial splits (Fig. 9). The amount of hæmorrhage from intratentorial vessels depends, therefore, on the position of the split and not on its degree. The relative absence of basilar subdural hæmorrhage, even in cases of complete bilateral tentorial splits, is another contrary indication to extensive bleeding from intratentorial vessels. Extravasated blood from this source would naturally gravitate into the infratentorial subdural space; here, however, blood is very seldom demonstrable at autopsy.

The frequent finding of blood in the subdural space surrounding the great cerebral vein or its tributaries is of great significance. This often represented the highest point of subdural hæmorrhage in the cranium. A stretching and tearing of these tributaries of the great cerebral vein is undoubtedly the usual source of hæmorrhage into the supratentorial fossæ. But owing to the minuteness of these tributaries in the newborn we were not able to demonstrate actual ruptured vessels. The great vein of Galen itself escaped in this series, although Holland has been able to demonstrate actual tears in several cases. In describing the mechanism he points out that the great vein of Galen extends between the fixed straight sinus and the relatively movable brain. In the process of molding, the vertex is raised and the vein of Galen is sharply kinked and stretched. This, apparently, will occlude the vein and often cause rupture of its tributaries.

Intradural hæmorrhage. This is a very common finding in the triangular space of the tentorium and is often associated with simply a stretching, or only superficial splitting of the tentorial fibers. In this series of cases, intradural hæmorrhage was a constant finding in definitely premature infants (Fig. 6). The only exception was Case 27. This was a dead-born fetus in which it is safe to assume that the intracranial lesions occurred after death. This intradural hæmorrhage is undoubtedly due to the greater friability of the dural septa and intradural vessels in the premature infant. The blood diffuses in a thin film between the layers of the tentorium cerebelli and falx cerebri. There is nothing to indicate that this hæmorrhage is of any clinical importance.

Tentorial splits. It seems more correct to refer to these tentorial lesions as splits rather than as tears. A careful gross and microscopic examination of the small superficial lesions, while of little clinical importance, will serve best to illustrate the true nature of the process. The gross appearance in such a case shows the

Fig. 6. Case 5. Premature fetus showing extensive superficial split of the stretched vertical fibers of the tentorium cerebelli. There is also an oval split in the anterior part of the falx cerebri. Note the diffuse intradural hæmorrhage. (Also illustrates method of opening skull.)

Fig. 7. Case 8. Complete split of the tentorium cerebelli with surrounding intradural hæmorrhage. The opposite side of this specimen is shown in Figure 8.

superficial fibers more prominent than usual, and an arrangement of these fibers into linear ridges. The separation of these superficial fibers is well demonstrated in the histological sections (Fig. 1). In the more extreme cases in which there is a complete split of both tentorial blades, a marked degree of stretching and separation of the fibers is readily demonstrable weli beyond the ragged margin of the split (Figs. 7 and 8). A similar picture of tentorial splitting may be produced artificially by exerting vertical traction on the vertex of the skull after the modified Beneke's exposure of the dural septa is made. The superficial vertical fibers, therefore, stretch, separate into bands, and finally split apart transversely (rather than tear), while the deep transverse fibers separate and finally split apart longitudinally.

Tentorial splits may be either unilateral or bilateral. The lesions are superficial, complete, or perforating in character. In the superficial type, there is an irregular transverse splitting of only the upper vertical blade of the tentorium. Thus the intact deep horizontal blade is exposed covering the cerebellum (Fig. 6). In the complete type both superficial vertical and deep horizontal blades are split (Figs. 7 and 8). When the anterior free margin remains intact the split is referred to as perforating in character (Fig. 9).

The frequency of tentorial splitting in premature infants after an easy spontaneous delivery is well recognized. There have been many explanations offered as to why the fibers of the tentorium cerebelli should be more prone to separate and rupture in the premature fetus than in the full term infant. Ehrenfest emphasizes the fact that the thinness of the premature infant's skull and the widely separated suture lines predispose to excessive molding of the fetal head during labor. With normal pelvic measurements one would expect the mechanical process of molding of the fetal head in its passage through the birth canal to be of shorter duration and less difficult in the premature than in the full term infant due to the smaller circumference of the fetal head. This was readily demonstrated in our cases. The average duration of labor in 11 premature infants was 10 hours (most of these were easy spontaneous deliveries), while the average duration in 19 full term infants was 20 hours. The majority of the latter group were finally terminated by operative interference. This evidence does not favor the mechanism of birth as the only important factor producing tentorial lesions in these premature infants. From a careful histological study of the tentorium cerebelli in these cases, we regard its cellular structure as responsible for its greater friability in the

Fig. 8. Case 8. A split of the anterior free margin of the tentorium with extensive stretching and separation of the fibers and intratentorial hæmorrhage.

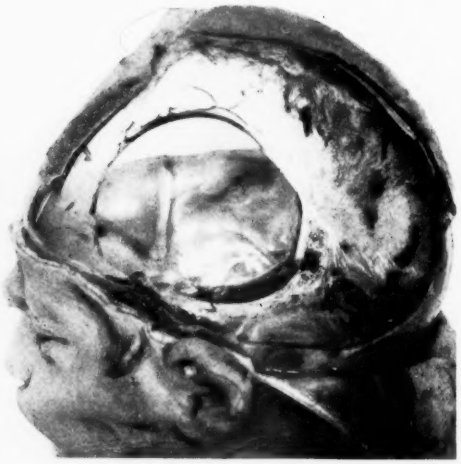

Fig. 9. Complete split of tentorium through the anterior free margin and a perforating split in the posterior fossa.

premature fetus. This opinion is based on a careful comparative study of histological sections taken from similar areas in the triangular tentorial space from the premature and full term infant. The sections from the premature infant show 3 important differential points (Figs. 2 and 3): (1) a predominance of fibroblasts with few collagen fibrils; (2) absence of elastic tissue fibrils (Wigert's stain); (3) relative abundance of lymph spaces and thin walled blood vessels. The immaturity of the fibrous connective tissue with its absence of collagen and elastic fibrils are all indicative of its increased friability.

The relative unimportance of tentorial splitting *per se* is well demonstrated by the specimen shown in Figure 10. This 5 month old infant never had symptoms referable to an intracranial lesion. He died of septicæmia following an exudative purulent mastoiditis and otitis media. There is gross evidence of scar tissue formation in the superficial tentorial blade and a vertical split close to the anterior free margin still remains open. A little yellowish brown pigment covered the dura mater over the posterior supratentorial fossa. This pigment gave the prussian blue reaction. It represented an old subdural hæmorrhage. Ford calls attention to the fact that as far back as 1890, Kundrat described old blood pigment in the meninges of children up to 9 months of age who gave no evidence of birth injury at the time of birth. The association of a partly healed tentorial lesion has been more recently described by Schule. Such cases are conclusive evidence that extensive tentorial splitting with subdural hæmorrhage heal spontaneously without symptoms.

Perforating split of the falx cerebri. This lesion was found in four instances (Cases 5, 7, 17, and 32). It will be noted (Fig. 6) that the long axis of the oval perforation is in the direction of the radiating fibers of the falx. The absence of intradural hæmorrhage and extravasated blood between the cerebral hemispheres indicates the avascularity of the anterior half of this dural septum. This was further demonstrated in the india ink injections. On account of the avascularity of the portion of the dura usually involved, these perforations in themselves are of no clinical importance. They are chiefly of interest because they are associated with extensive bilateral tentorial splitting. This was complete in three instances and superficial in the premature fetus shown in the photograph.

ETIOLOGY

Subdural hæmorrhage with a splitting of the fibers of the tentorium cerebelli are indisputable evidence of intracranial trauma. The

causes of intracranial trauma are conveniently divided into predisposing and determining factors. By far the most important predisposing factor to intracranial hæmorrhage as well as to tentorial splitting is prematurity. The anatomical explanation for both these lesions in the premature fetus has already been indicated. The 13 fetuses in this series of 32 cases represent a frequency of 40 per cent.

Syphilis is apparently important only as a predisposing cause of tentorial splitting in so far as it tends to favor premature birth of the fetus (Brissaud).

The mechanical determining factors are by far the most important of the determining causes of these intracranial lesions. Practically without exception lesions of the dura mater are partly due to mechanical causes. Beneke (2) described 14 cases of tentorial tears in 100 autopsies on stillborn babies. Two years later Seitz reported tentorial lacerations in half of his cases. More recently Capon investigated 80 neonatal and stillborn infants. He found intracranial hæmorrhage in 37.5 per cent and dural lacerations in 47.5 per cent. In a series of 145 autopsies on babies under 4 weeks old, we have found intracranial hæmorrhage in 50 cases. This represents a frequency of 37 per cent. In 32 of these 50 cases, the hæmorrhage was very largely subdural and there was also a splitting of the fibers of the tentorium cerebelli. This represents 64 per cent. In other words, about two-thirds of our cases of subdural hæmorrhage showed a split tentorium. Many of these were superficial and small, however, and would have been overlooked in a less careful routine examination.

It is not within the scope of this paper to discuss the forces of stress and strain on the fetal head during its passage through the birth canal. It is well recognized that excessive head molding, which accounts for a large number of these lesions, is based on a disproportion in size between the maternal bony pelvis and the fetal head. Beneke's (2) original work on this subject has more recently been elaborated by Holland, Ehrenfest, Warwick, and others. Holland has shown quite convincingly that pressure of the fetal head in one diameter results in a shortening in that diameter and a compensatory increase in some other diameter. Greenwood agrees that the vertical diameter of the head is always increased in breech presentation, but he shows by careful measurements with plaster casts that the suboccipital bregmatic diameter, which is engaged in normal labor, does not produce vertical elongation of the head but causes an actual decrease in this diameter with an increase in the horizontal component. This seems a most logical explanation for the much larger proportion of tentorial splitting in brow and breech presentations over vertex presentations. Ford points out the interesting observation made by Schwartz that animals with solid skulls do not suffer birth injuries.

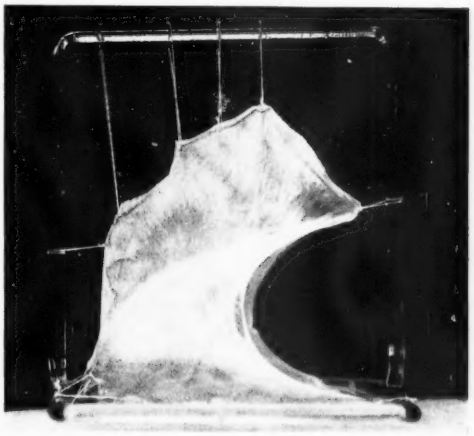

Fig. 10. A partly healed tentorial split. Note the persisting small oval perforation just behind the anterior free margin.

There can be no doubt that the mechanical process of head-molding during labor, which is so often responsible for intracranial trauma, is in many instances further aggravated by the operative manipulations of even the most careful obstetrician. In this series of 32 cases exactly half of them have a history of operative interference. This figure indicates that every case of prolonged and difficult labor must be treated as a potential case of subdural hæmorrhage with split tentorium cerebelli. Any operative interference, however slight, must be done with extreme care, on the general assumption that intracranial trauma has already occurred.

CLINICAL AND ANATOMICAL ANALYSIS OF SUBDURAL HÆMORRHAGE WITH SPLIT TENTORIUM CEREBELLI

Case No.	Intra-uterine age	Neonatal age	Para	Duration of labor in hours	Position at onset of labor	Course of labor and indication for operative interference	Length in cm.	Weight in grams	Tentorial split				Subdural hæmorrhage				Other gross lesions
									Right		Left		Position		Degree		
									Extent	Length in cm.	Extent	Length in cm.	Right	Left	Right	Left	
1	Term	Dead-born	I	10	L.O.A.	Low forceps with gentle traction. Eclampsia	48	2,680			Superficial	1.5		Posterior fossa		Small	
2	Term	9 hr.	I	10	L.O.A.	Spontaneous delivery	52	3,230			Superficial	0.8		Post. and middle fossæ		Small	
3	Term	6 da.	VII	27	L.O.A.	Four loops of cord about neck. Difficult spontaneous delivery	50	3,470	Complete	2.0			Post. and middle fossæ		Large		
4	Term	9 da.	V	c.a. 13	L.O.A.	Indefinite onset of labor. Slow spontaneous delivery	50	2,450	Superficial perforating	0.5							
5	8½M	20min.	III	c.a. 16	L.O.A.	Spontaneous delivery	47	2,830	Superficial	1.2	Superficial	1.5	Post. and middle fossæ	Post. and middle fossæ	Small	Small	Split in anterior third falx cerebri 3 by 2 cm.
6	6 M	Dead-born	I	8	R.S.A.	Bag induction. Eclampsia	36	1,870	Superficial	1.0	Complete	1.6	Post. and middle fossæ	Post. and middle fossæ	Moderate	Moderate	
7	8 M	30min.	II	c.a. 8	R.S.A.	Easy spontaneous delivery	48	2,110	Complete	0.8	Complete	0.8	All fossæ	All fossæ	Large	Large	Split in middle third falx cerebri 3 by 1.6 cm.
8	Term	26 hr.	III	10	R.O.P.	Cervix not fully dilated. Podalic version with extraction. Prolapsed cord	50	3,190	Complete	0.5	Complete	1.5	Post. and middle fossæ	Post. and middle fossæ	Moderate	Moderate	
9	Term	13 da.	II	c.a. 48	L.O.P.	Midforceps. Transverse arrest of head	52	2,820			Superficial	1.2		Post. and middle fossæ		Moderate	
10	8½M	20 hr.	II		L.O.A.	Spontaneous delivery	50	2,400			Superficial	1.0					Intratentorial hæmorrhage bilateral.
11	8 M	6 da.	I	6	L.O.A.	First twin. Difficult low forceps. Flat pelvis	45	1,550			Superficial	1.2		Post. fossa		Small	
12	Term	Still-born	VIII	c.a. 20	R.O.A.	Slow spontaneous delivery	50	3,000	Complete	0.3	Complete	1.8	Post. and middle fossæ	All fossæ	Small	Large	
13	7 M	19 hr.	VI	21	R.O.A.	Podalic version and extraction. Transverse position. Prolapsed arm	43	1,950	Complete	1.0	Complete	1.4	All fossæ	All fossæ	Large	Large	
14	6½M	Still-born				Spontaneous delivery. Uræmia	37	1,510	Superficial complete	0.6	Complete perforating	1.0	Post. fossa	Post. and middle fossæ	Small	Moderate	
15	Term	4 da.	III	9	L.O.A.	Low forceps. Rigid perineum	50	2,660			Superficial	1.6		Post. fossa		Small	
16	Term	Still-born	I	c.a. 29	R.O.A.	Slow spontaneous delivery	50	3,350	Complete	1.2	Complete	0.8	Post. fossa	Post. and middle fossæ	Small	Moderate	

17	7½M	1 hr.	I	20 min.	L.O.A.	Bag induction. Midforceps. Eclampsia	42	2,160			Complete perforating	1.5	All fossæ	All fossæ	Large	Large	Perforation into torcular herophili left side. Split in middle third falx cerebri 4 by 2.3 cm.
18	Term	4 da.	I	29	L.O.A.	Low forceps. Rigid perineum. Eclampsia	50	3,160	Complete	0.7	Superficial	1.8	All fossæ	Post. and middle fossæ	Large	Small	
19	7½M	4 hr.	IV	6		Placenta prævia marginalis. Contracted pelvis. Dystocia due to scar tissue. Cæsarean section	46	2,260	Superficial	0.8	Superficial	1.2					Intratentorial hæmorrhage bilateral.
20	7½M	Dead-born	I	19	R.O.A.	Spontaneous delivery	46	1,480	Superficial	2.5			Post. and middle fossæ		Small		
21	Term	Still-born	II	15	R.O.A.	Spontaneous delivery	48	1,580			Complete	2.2		Post. and middle fossæ		Small	
22	7 M	18 hr.	V	5	R.O.A.	Second twin. Spontaneous delivery	43	1,640	Complete	2.0			All fossæ	Middle fossa	Large		Fetal hydrops.
23	8 M	Still-born	III	8	R.O.A.	Placenta prævia marginalis. Bag induction. Midforceps	46	2,750	Complete	2.5	Complete	1.5	Middle fossa	Middle fossa	Small	Small	
24	Term	Dead-born	II	12½	L.O.P.	Contracted pelvis. High forceps. Trauma of labor	50	3,850	Complete	2.8	Complete	3.0	All fossæ	All fossæ	Large	Large	
25	Term	Still-born	I	58	L.O.A.	High forceps. Persistent oblique. Rigid perineum	52	4,300	Complete	1.4	Complete	1.6	All fossæ	All fossæ	Large	Large	Infarction adrenal bilateral.
26	Term	3 da.	VI	14½	R.O.A.	Spontaneous delivery	50	3,060	Complete	1.0	Complete	1.2	All fossæ	All fossæ	Large	Large	
27	c.a. 7 M	Dead-born	II	8	L.O.A.	Spontaneous delivery	44	1,800	Complete	2.2	Complete	2.2					(Postmortem lesion.)
28	Term	5 da.	I	c.a. 14	L.S.A.	Difficult delivery of large head. Manipulation	42	2,840	Superficial	2.6	Superficial	0.5	Post. and middle fossæ	Post. and middle fossæ	Small	Small	Infarction right adrenal.
29	Term	7 hr.	III	7	R.O.A.	Podalic version and extraction. Contracted pelvis. Floating head	50	3,170	Superficial	1.5	Complete	1.5	All fossæ	All fossæ	Large	Large	Large subdural basilar hæmorrhage.
30	Term	6 hr.	III	6	L.O.A.	High forceps. Persistent right transverse	52	3,420	Complete	1.8	Complete	2.8	Post. and middle fossæ	Post. and middle fossæ	Moderate	Moderate	
31	Term	2 da.	I	c.a. 40	R.S.A.	Difficult spontaneous delivery. Justominor pelvis	46	2,400	Superficial	1.2	Superficial	1.0	Post. fossa	Post. and middle fossæ	Small	Small	Congenital anomaly of heart. Tetralogy of Fallot
32	Term	30 hr.	V	10	R.O.P.	Podalic version and extraction. Flat pelvis. Floating head	50	1,000	Complete	2.4	Complete	2.0	Post. and middle fossæ	Post. and middle fossæ	Moderate	Small	Split in middle third falx cerebri 5 by 3.5 cm.

The application of forceps is by far the most common operative manipulation associated with these intracranial lesions. It represents 31 per cent of our series of cases.

Podalic version and breech extraction is next in order of frequency. Combined with four primary spontaneous breech deliveries, this group represents a total of 8 cases, or a frequency of 25 per cent. The mechanical explanation for the well recognized frequency of tentorial splitting in breech presentations was first advanced by Wilke and Seitz in 1912. More recently Greenwood has shown that the vertical lengthening of the head is usually due to the occiput being firmly pressed against the symphysis pubis. Crothers has demonstrated that the vertical lengthening of the supratentorial chamber in these cases causes a marked strain and stretching of the tentorium. This strain is further increased by any traction on the body from below which he thinks is partly transmitted through the vertebral column and spinal cord.

The more unusual measures which may be associated with subdural hæmorrhage and tentorial splitting include cæsarean section operations and some methods of resuscitation. A cæsarean section was the operative interference in Case 19. There were two indications: a contracted pelvis and a placenta prævia. This woman had a uterine dystocia which was probably due to three previous cæsarean sections. The operation was done after slight engagement of the head in the left occiput anterior position for 6 hours. The baby was definitely premature. This case is of particular interest because it suggests two possible causes of subdural hæmorrhage and tentorial splitting which would be even more liable to occur in prematurity: (1) early engagement of the head in a small pelvis, and (2) a forced extraction of the head through a uterine incision of insufficient length.

Since Bauereisen's experiments demonstrating that tentorial splits can be produced by the lateral compression of the head by holding it fixed during Schultze's method of resuscitation, this method has been generally discarded. It was not employed in any of this series of cases. Any method of resuscitation which necessitates compression of the head, or swinging methods which cause engorgement of possibly ruptured intracranial vessels, is obviously contra-indicated.

ANALYSIS OF CASES

Clinical analysis. The cases fall naturally into three groups depending on the type of labor and the necessity for operative interference.

	Cases
Group 1. Easy spontaneous deliveries	10
Group 2. Difficult spontaneous deliveries without operative interference	6
Group 3. Difficult labors with operative interference	16

Group 1. Prematurity was the common predisposing factor to the intracranial lesion.

Group 2. In these 6 cases (3, 4, 12, 16, 28, and 31) of slow and difficult but spontaneous full term deliveries, the mechanical process of molding of the fetal head in its passage through the birth canal for some reason was unduly prolonged. This group indicates that the factors which are responsible for prolonged and difficult labor in the full term infant are just as important causative factors to tentorial splitting and subdural hæmorrhage as are the operative manipulations of the experienced obstetrician.

Group 3. The 16 cases of difficult labor with operative interference may be grouped according to type of operative procedure used.

	Cases
Forceps deliveries	10
Podalic version and breech extraction	4
Bag induction spontaneous breech	1
Cæsarean section	1

The indications for operative interference were always very definite. They are noted in the table accompanying this article.

Anatomical analysis. The intracranial anatomical lesions are grouped as follows:

Summary of tentorial splits:

	Cases
Unilateral superficial	8
Unilateral complete	4
Bilateral superficial	4
Bilateral complete	12

A summary of subdural hæmorrhage in relation to tentorial splits is shown in the following table.

SUBDURAL HÆMORRHAGE AND TENTORIAL SPLITS

Type of tentorial split	Degree of subdural hæmorrhage				
	Cases	Small	Moderate	Large	Absent
Unilateral superficial	8	5	1		2
Unilateral complete	4	1		3	
Bilateral superficial	4	3			1
Bilateral complete	12	1	4	6	1
Bilateral mixed	4		2	2	

There is obviously no absolute relation between the extent of the tentorial damage and the amount of subdural hæmorrhage. It is notable, however, that large hæmorrhages are found most frequently in cases in which there is a complete bilateral tentorial split.

SYMPTOMS

Only a short reference to the antemortem symptoms will be included here. The typical textbook picture of intracranial trauma is seldom recorded in the histories of these babies. Shallow and labored respirations with usually a persistent moderate degree of cyanosis are by far the most constant clinical findings mentioned. In over 90 per cent of the neonatal infants in this series, these are the only recorded symptoms. The remaining cases showed, in addition to this picture of asphyxiation, the following signs of intracranial irritation in order of their frequency: bulging of the anterior fontanelle, convulsions, and spasmodic twitching of both extremities and face. All those cases which had these latter signs showed a large or moderate subdural hæmorrhage in the supratentorial fossæ on one or both sides.

It is hoped that this anatomical analysis of the cases here recorded may, in addition to its theoretical interest, suggest to neurological surgeons means and methods to lower the high mortality in this type of intracranial injury.

SUMMARY

Based on observations from an analysis of 32 cases of subdural hæmorrhage with tentorial splitting, our conclusions are:

1. Subdural hæmorrhage is *the* important intracranial lesion in most cases of birth trauma.

2. There is nothing to indicate that intradural hæmorrhage or tentorial splits *per se* are of noteworthy clinical significance.

3. The subdural hæmorrhage is largely supratentorial and often bilateral. It is usually due to a stretching and rupturing of the small tributaries of the great cerebral vein near its junction with the straight sinus.

4. Tentorial splits are relatively more numerous in the premature than in the full term infant, partly because of the greater immaturity of fibers of the dural septa in prematurity.

5. The causes of prolonged and difficult labor may be equally as important in these intracranial lesions as the operative interference.

6. Signs of asphyxiation were constant, but definite signs of intracranial irritation were recorded in only a small minority of cases.

I wish to express grateful thanks to Mr. J. Giroux, of the McGill University Museum Staff, for the valuable technical help which has made this investigation possible.

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SENILITY OF BONE AND ITS RELATION TO BONE REPAIR

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DURING the preparation of material for a previous article on "The Organic Content of Bone,"[1] the variation in thickness and the gross appearance of the walls of the femur, tibia, and fibula were so marked as to be considered worthy of investigation. Sections of these bones were ground and studied and extensive changes were noted in some of them. The thought naturally arose that here might be the solution of the variation in the repair of bone in elderly individuals.

The practitioner is familiar with the fact that in those past the middle period of life, fracture of the femur or of the tibia may be a serious matter, as far as future locomotion of the patient is concerned. In some individuals sudden excessive muscle strain may be attended with near disastrous results. The physician is also acquainted with the fact that in the aged a mere stumbling or tripping, with the attendant sudden and violent muscle contraction, will cause the femur to snap. Why? Also, such a patient may have a prolonged or protracted recovery, or, not unlikely, may be bedridden for the remainder of his or her days, or, at best, hobble around with the aid of a cane or crutches. Why?

On the other hand, such individuals may have a complete and uneventful recovery in the usual time and show no untoward effects of the accident. Why? We are as old as our bones may be said of these structures as well as of the various organs of our bodies. There must be some physical or chemical basis for this difference and this should show microscopically. With this in mind, a careful study of many sections of ground bone was made to determine the presence of such a condition.

In order to understand the change that may occur in the bones of the aged, it will be necessary first to consider the structure of compact bone. One would naturally believe that compact bone would be the same, no matter where found and at any age. One would also be inclined to believe that compact bone would be the same in all animals possessing a pronounced skeleton. However, these suppositions are far from the truth. In fact, there is a difference in the structure of the compact bone, say the femur, in the various classes of the human race (negro, yellow-brown, and white). This type of bone shows evolutionary changes that are not as simple as they may seem. The lowest and earliest form is basic bone and this advances to the lamellar variety; then laminæ form, and ultimately, by the addition or formation of haversian systems, the highest type is reached. As in the case of civilization, the penalty of which, we may say, is disease, so in the evolution of bone in the development of a highly evolved type, in which the haversian systems predominate, there is likewise a penalty attached, that of the peculiar changes of senility. This seemingly affects only the highest type of bone, that of the Caucasian, in which the haversian systems have reached their greatest development.

In order better to follow the description of the various types of compact bone, a few of the terms used will be elucidated.

Basic bone is the earliest form of osseous tissue and consists of only moderately differentiated bony substance, as is seen in early fetal life. Lamella formation has as yet not occurred. Basic bone represents the first end-product of the replacement of fibrous tissue or cartilage by osseous tissue.

A *lamella* is a thin layer of osseous tissue and represents a comparatively small structure of the whole.

A *lamina* is larger and more complex; it consists of a set of bone layers or lamellæ. It comprises a variable number of concentric sheaths of lamellæ just under the periosteum or bounding the medullary cavity, each lamina being separated from the adjacent one by systems of plexuses of vascular canals that extend parallel to the marrow cavity. Laminæ are seen in birds, mammals, in later fetal life, and in early childhood.

[1]Anat. Record, 1921, xxi, May.

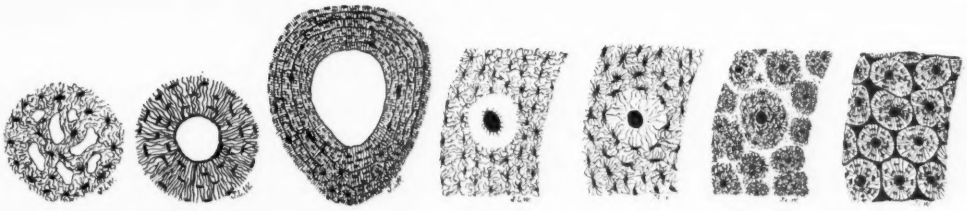

Fig. 1. Fig. 2. Fig. 3. Fig. 4A. Fig. 4B. Fig. 4C. Fig. 4D.

Fig. 1. Basic or undifferentiated bone.
Fig. 2. Differentiated bone with concentric lamellæ.
Fig. 3. Latest stage of differentiation of laminæ.
Fig. 4. Various stages in the progressive development of an haversian system: *A*, Early (amphibian); *B*, later (reptile); *C*, third stage; the systems are more distinct though still hazy (birds); *D*, completely differentiated systems (mammals and Man).

An *haversian system* consists of a central canal surrounded by three to eight concentric lamellæ; between the latter are the lacunæ and the canaliculi that usually have a straight course. The canaliculi connect the lacunæ of a system to one another. In the early form of bone, the lamellæ are not concentrically arranged, are more or less independent of the canals, and are variable in diameter. The lowest type of haversian systems is seen in the amphibians, where they are rather hazy and indistinct. In reptiles and birds they become more distinct, while in man they reach their climax of development.

TYPES OF BONE

While there are three main types of compact bone, these do not commonly exist individually or separately. The compact bones of the various animals are chiefly combinations of any two or of all three types.

Type I. Basic bone represents the first type and this is the earliest form of osseous tissue. It is only moderately differentiated bony substance and is seen in early fetal life. The lamellæ have as yet not been differentiated. This state may be followed by lamellæ formation of the concentric type and the lamellæ then become concentrically arranged. At first the lacunæ are round, then oval, and lastly they become long and narrow. In many animals the changes stop here and so they belong to the first type. The bone lamellæ are joined by cement, but the exact manner in which this and the lamellæ are formed is unknown. Although many animals possess this simple type, it is not common. Subtypes exist, but they will not be considered here.

Type II. In this form, the lamellæ have become somewhat differentiated and haversian systems may even be present, but the ordinary lamellæ predominate. If the differentiation is not extensive, it is spoken of as incomplete, as in some birds. Here the basic bone is separated, partially, into distinct lamina by a few short vascular canals that have a general concentric position.

In the complete variety, the vascular canals form a more or less complete circuit, the laminæ are well defined, the lamellæ are completely developed, and the lacunæ are long and narrow. This represents lamellar and laminar bone.

Type III. In this variety the haversian systems are very numerous and reach their highest development. They represent the unit of structure. The internal and external lamellæ are merely thin bands of bone. The stage of differentiation may be complete or incomplete.

In the incomplete form, there may be only a slight suggestion of haversian canals and systems; the canals are minute, irregular, and directed parallel to the long axis of the bone. The canal is surrounded by a clear zone of bony substance, usually circular in outline; the canaliculi that extend to the neighboring lacunæ may readily be noted. The lamellæ are not concentrically arranged and are more or less independent of the canal. This structure is seen in amphibians and up to Man.

A somewhat more advanced stage shows the haversian systems fairly well outlined,

Fig. 5. Medullary index. *A*, Femur showing a medullary index of 31.7 per cent (a little below average). *B*, Tibia of same extremity showing a medullary index of 20 per cent (probably quite low). Bone normal.

and a more definite relationship between the lacunæ and the haversian system and canal is noted. This is first found in the reptilian class but is not seen in birds.

In another step of differentiation, the canaliculi are intricate, the haversian lamellæ are regularly and definitely arranged, concentric to the canals, and the canaliculi form a dense plexus. The complete system in its earliest form has a somewhat dull and hazy appearance and is found only in birds.

In the completely differentiated variety, the haversian systems are clear and distinct and this type is found in higher mammals and Man. It does not occur in the lower mammals, such as the monotremes, marsupials, edentates, and chiropters. Haversian systems, therefore, are found in nearly all types of bone, but in varying degrees of development, from the mere outline of the amphibians to the sharp, clear, and definite type in Man.

Types I and II of compact bone are chiefly combinations of lamellæ and haversian systems. Types I, II, and III are combinations of haversian systems, lamellæ, and laminæ.

MEDULLARY INDEX

An anatomical factor bearing upon the strength of compact bone and its repair, is the *medullary index*. An increase in the index will mitigate against a satisfactory resistance to bone strain and also against its successful repair in the event of fracture. This is due to the fact that the usual causative factor of increase is senile change of bone. This itself reduces the quality and strength of the bone, thereby greatly influencing the reparative process unfavorably.

The medullary index represents the relation between the diameter of the bone and the diameter of the medullary cavity. It really indicates the thickness of the bony shell or wall. It has been worked out in the femur, but not in the other long bones, except the tibia in the present work. The thicker the bony wall, the stronger the bone will be; the marrow cavity will be smaller and, therefore, the medullary index lower. As the bony wall becomes thinner and the marrow cavity larger, the index increases. This naturally has its effect upon the resistance of the bone as well as upon its repair.

The medullary index is lowest in reptiles (in turtles there is practically no marrow cavity) and is given as 26.1 per cent. In birds, the index is highest with an average of 159.0 per cent. The average index in the human is 38.6 per cent, varying from 35.9 per cent in the Caucasian to 41.9 per cent in the negro. In the yellow-brown race the index is 39 per cent.

BONE REPAIR AND REGENERATION

When a bone is fractured and it heals with the formation of normal bone, this process constitutes bone repair. It is in reality, bone regeneration. Only a few of our tissues have the power of regeneration, but fortunately we are capable of regenerating osseous tissue. In those individuals in whom bone regeneration is low or wanting, the union of the fractured parts is by means of fibrous tissue or cartilage. With the attendant loss of the original rigidity, the use of the limb is greatly affected or lost.

Bone readily regenerates, provided the periosteum is present. If a bone be broken, a blood clot forms in the marrow cavity beneath the periosteum and between the ends of the fragments. The periosteum is rarely torn completely through, as there is usually a bridge of membrane between the ends of the fragments of the bone.

The clot becomes organized through the proliferation of the various cells, especially those of the endothelial type, and fibroblasts are formed. This fibrous mass is invaded by

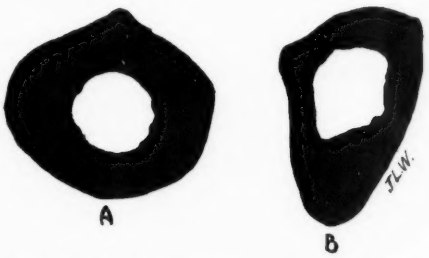

Fig. 6. Medullary index. *A*, Femur showing medullary index of 26.7 per cent (rather low). The bony wall is quite thick. *B*, Corresponding tibia showing a medullary index of 44.2 per cent (probably about average). Bone normal.

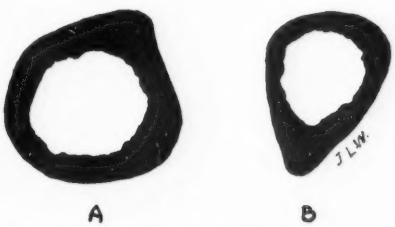

Fig. 7. Medullary index. *A*, Femur showing a medullary index of 62 per cent (very high). *B*, Corresponding tibia showing a medullary index of 72 per cent (very high). Both bones showed evidence of extensive senile changes.

osteoblasts from the marrow and the deeper parts of the periosteum and these cells proliferate. This fibroblastic mass then becomes vascularized and constitutes *granulation tissue*. Following this, bone may be directly formed or a moderate amount of cartilage may first be produced. This represents *callus* and that portion in relation with the periosteum is likely to show some cartilage formation, but the remainder usually becomes directly ossified. An absence of the osteoblasts leave the mass fibrous or cartilaginous. This may be a permanent state and is presumably the result of weakened activity upon the part of the osteoblasts. When the fibrous mass holding the fragments of bone together, becomes ossified, then the bony union is completed. This is all a replacement process and not a change of one kind of tissue into another.

One might state that during the first week after the fracture, there is the formation of the clot and the organization thereof; during the second week this becomes greatly strengthened by dense fibrous tissue in and beneath the periosteum. During the third week ossification begins at points farthest from the ends of the fragments; usually by the end of the sixth week ossification between the ends of the fragments is completed. This intervening new bone is a solid, spindle-shaped mass that fills the marrow cavity as well as projects beyond the general surface of the bone.

The name *callus* is successively applied to the fibroblastic tissue, the granulation tissue, the fibrous tissue, and the resultant bone. That portion in the marrow cavity is the central callus; that surrounding the bone beyond the general level of the bony surface is the ensheathing callus. These two types constitute the temporary callus. The central callus may later be removed, thus restoring the marrow cavity, while the osteoclasts tend to smooth the greater irregularities of the surface of the new bone.

Bast and Sullivan have recently completed some experimental work on the repair of bone. They call the osteogenetic layer of the periosteum the "cambium layer." They also refer to two types of bone dissolution, i.e., with and without the presence of osteoclasts.

The osteogenetic cells that line the marrow surface of the bone, those lining the haversian canals, and those constituting the cambium layer are the most important in the repair of bone. The cambium layer exhibits the most important changes during the beginning of the repair process. During the second day, already the cambium layer becomes thickened through the multiplication of its cells. At first the cells are of the simple squam-like type. These later become cuboidal and then stratified to six or more layers. Spicules of bone are next formed in this cambium on the underlying bone. These changes involve one-eighth to one-quarter of the circumference of the bone on each side of the saw cut. If the cambium is injured much in the cutting, then the callus is usually formed by the uninjured cambium. The formation of the callus is rapid during the fourth to the fifteenth day. Over the ends of the cuts the formation continues until the eighteenth day. The enlarged cambium farthest from the cut becomes reduced as early as the ninth day when

osteoclasts begin to absorb the newly-formed callus immediately beneath it. This process of solution continues toward the cut until the eighteenth or nineteenth day.

The periosteum seems to strip very readily and separates into two layers, the fibrous portion that is quite thick and a thin, cellular portion that remains attached to the bony surface. The stripped periosteum will not regenerate bone directly but may produce cartilage that is then replaced by bone. If the periosteum is removed so as to include the cellular layer, then this membrane will regenerate bone directly therefrom.

Cartilage callus did not form in these experiments, but Wieder described marked cartilage calluses in complete fracture experiments, but an absence thereof in saw experiments. The cartilage masses found in the present experiments were not large and were independent of the bony callus. They were found in the young connective tissue at the cut ends of the fibrous periosteum outside of the cambium. They never bridged the cuts. At about the seventh to the ninth day osteogenic buds from the underlying callus invaded these cartilage masses and replaced them, reducing them rapidly.

It seems as though osteoclasis is practically the sole method by means of which the external callus is removed and excavations in the old bone are made. In a few instances "bone dissolution" was noted in the large masses of the external callus. In removal of internal callus the osteoclasts play a part, but where the internal callus is large, bone dissolution seems to be the method of removal. This has the appearance of fatty and mucoid degeneration and is noted as early as the ninth day. It usually takes place in the center of the bony spicules at a time when the osteogenetic cells still appear active on the surface. It is possible that a vascular or nutritive disturbance at the center of the spicules may be the initial cause of this type of bone destruction.

These writers conclude that the cambium layer, the osteogenetic cells lining the haversian canals, and the periosteum are all active in the formation of the calluses that reunite the old bone at the line of fracture.

The union of the severed bone is strengthened by means of a sort of dove-tailed union made possible by the extension of the new bone forming in the cut into the excavated spaces of the adjacent old bone.

The reduction of the external callus is accomplished by osteoclasis.

Cartilage when present arises from the connective tissue cells at the cut ends of the fibrous periosteum.

In the elderly, however, the story is somewhat different. If the fracture is of the shaft of the bone, repair usually takes twice as long as in those in the prime of life, and the results are not what they would be at an earlier age. If the fracture occurs at the neck of a bone, as a rule it does not heal at all, but remains in an un-united condition.

SENILITY OF BONE

As previously stated, no attempt at a scientific explanation has been made in those cases of failure of repair of fracture in the elderly individuals. The fact that all repair processes are usually much slower and poorer in the elderly, has sufficed and no attempt has been made to find a better explanation. It has been looked upon as a condition that we must accept as a companion to old age, with some individuals subject to it and others immune.

Also no attention has been paid to the difference in structure of compact bone at different ages and in different individuals. No one has suggested that there may be physiological or histological reasons for the differences in reparative processes in connection with bone fracture. It apparently has been taken for granted that compact bone is compact bone ever and always and that no definite changes take place in it from the adult period to old age. At least no mention is made thereof and no stress is laid thereon.

But compact bone is not always and ever the same at different ages no more than a kidney at 20 years is necessarily the same as at 40 or 60 years of age, and while a defective bone may not exhibit its defects as readily pronouncedly nor as vitally as a defective kidney or heart, nevertheless, the defect manifests itself under certain conditions.

Compact bone apparently undergoes senile change. This peculiar alteration seems to concern only the higher mammals, the femora of the amphibians, reptiles, birds, and bats do not show these changes. They have been found in only a few of the mammals. The majority of femora of Caucasians seems to be subject to this change, irrespective of age. The process may begin at 25 years or may not be present at 80 years.

The changes are limited primarily to the haversian systems; secondarily the interstitial lamellæ suffer. The haversian systems seem to be the last developed, phylogenetically and so seem to be the weakest parts of the bone.

The lamellæ of the system affected become darker from the deposition of granules of an organic nature; these intensify the clearness of the serrated edges of the lamellæ and the cement between them. The granules increase in number, involving lamella after lamella until the whole system is opaque and black. The lamellæ gradually disintegrate into amorphous granules that drop into the haversian canal and are then removed by the blood vascular (?) or lymph vascular (?) systems. As the process progresses, the interstitial lamellæ are secondarily affected and a cavity is thus formed. The lamellæ of the adjacent haversian systems undergo like changes and the resultant cavities coalesce to form a few larger ones. These changes continue with greater or less rapidity and the bone thereby becomes reduced in diameter (?), weight, and strength. At times it becomes a mere shell. The process starts in those lamellæ nearest the medullary canal and proceeds from within, outward. These changes are most numerous in the outer and anterior walls of the middle portion of the shaft (Foote).[1] In other portions of the shaft the canals and systems will be seen to be in good condition.

A careful study of the various sections shows that this peculiar change starts in isolated haversian systems and proceeds until all of the lamellæ of the system are destroyed. As the change progresses, the interstitial lamellæ between several haversian systems fall victim to the process, and with their removal the several isolated areas of destruction merge into one large one. The changes will be given as they occur in the two bones studied, the femur and the tibia.

[1]A contribution to the comparative histology of the femur. Smithsonian Institute, 1916.

FEMUR

In the early stages the alterations are confined to the vicinity of the marrow cavity. The ordinary unaffected canals measure about 30 to 40 microns in diameter. Those that have been attacked by this condition measure from 160 by 195 microns to 170 by 310 microns. It seems that interstitial lamellæ also may have been destroyed as the cavities are not regular in form. In such sections there are very few such areas near the periosteal surface of the bone.

As the change progresses, the riddling change becomes manifest to the unaided eye. The enlarged canals are mostly anterior and spread toward the periphery. The largest are in the vicinity of the marrow cavity and measure from 200 by 220 microns to 275 by 375 microns.

In advanced stages the bony shell is thinner and is quite porous as it becomes riddled by these enlarged canals. These extend through at least one-half of the thickness of the remaining bone, while in the thickest part of this bony shell these enlarged canals are scattered through to the periosteal surface. The thinning of the bony shell is due to the fact that these enlarged canals around the marrow cavity have become confluent with one another and also with the marrow cavity, thus enlarging the latter and making the bony shell thinner. In the sections showing the greatest changes the bony shell is quite thin. The enlarging canals are noted most numerous in the region of greatest thinness of the bony shell.

TIBIA

In the early stages, the unchanged canals measure from 30 to 40 microns in diameter. The enlarging canals are in the vicinity of the marrow cavity and measure from 110 by 170 microns to 170 by 210 microns, being somewhat irregular in shape.

As the process progresses, it becomes more readily noted with the unaided eye. While the

anterior margin is not noticeably decreased in thickness, there are many enlarged canals near the marrow cavity and then others scattered through to the periosteal surface. The lateral wall is somewhat thinner than usual and there are many enlarged canals extending throughout the entire anterior wall. Apparently one group formed a line to the periosteal surface weakening the bone, thereby causing it to break at this point during grinding.

In the advanced stage the entire bony shell is thinner and the areas of the destruction are readily noted with the unaided eye.

Figure 8 shows an example of extensive alteration. The marrow cavity is shown at *M*, but the many enlarged canals are as yet not in continuity therewith. One can see the many normal canals and haversian systems throughout the section, on the one hand, and several enlarging canals (in the early stage) at *a*,*a*, on the other hand. The numerous large irregular areas *c*,*c*, are produced by the destruction of the interstitial lamellæ between several destroyed systems causing these cavities to become confluent.

One can readily appreciate the condition when those spaces near the marrow cavity become connected therewith. The marrow cavity becomes larger and the bony wall thinner, weaker, and less resistant to strain. As these areas form in the remainder of the bony wall, it becomes porous and fragile.

The changes may be summarized as follows:

1. Dissociation of the organic and inorganic constituents of the haversian lamellæ.
2. The appearance (deposition?) of granules in the lamellæ.
3. Extensions of the deposits to the periphery of the system.
4. Absorption and disappearance of the lamellæ from within, outward.
5. Widening of the canals and thinning of the system.
6. Disappearance of the haversian systems and the formation of irregular spaces.
7. Decrease of diameter (?), thickness of bony wall, weight and strength of the bone.
8. Increased medullary index.

At the onset these senile changes show only in the haversian systems. Why they should start here and not elsewhere is not definitely known. These changes may be connected with the blood supply variation or may be due to a variability in the chemical stability of the bony substance or to both. The blood supply might be affected by arteriosclerotic changes in the blood vessels. The circulation is more complicated in the third type of bone and so it would more readily be subject to structural variation. Also those structures developed last phylogenetically are usually weakest; this fact may explain why they undergo these changes.

SENILITY OF BONE IN RELATION TO BONE REPAIR

We are familiar with the fact that bones are usually united by the end of the sixth week after the fracture, all things being normal. The patient begins the use of the upper extremity by that time and is merely cautioned against excessive muscle strains and muscle actions. With the lower extremity it is different. The patient does not make use thereof so soon. The fragments are, no doubt, united by that time, but they are not yet able to bear the full weight of the body; this must be done gradually.

We are also familiar with the fact that sometimes bones are united by a cartilaginous union and when the part is used, it exhibits what is called a false joint. In other words, very little osseous tissue may form in the callus. Is this due to mechanical disturbance of the parts, faulty nutrition, or is it a disturbed endocrine state or a condition due to a faulty metabolism? It would seem that, in some instances, it is due to the latter condition as that type of union is sometimes seen in alcoholics. Also in those individuals who cannot lie still, incomplete ossification will occur. In other words, in most cases, unless the parts are maintained in a continuous approximate state, bone formation will not be complete. The irritation due to the movements of the fragments, seems to mitigate against perfect ossification.

In the aged it is known that a mere stumble or tripping with the attendant sudden and violent muscle action may cause a femur to snap. Why? Also such patients may have a prolonged or protracted recovery and not unlikely be bedridden for the remainder of their

days. Why? Other elderly individuals may have a complete and uneventful recovery. The bones grow old just as other organs of the body may do and usually do.

Upon examining some of the ground sections it is not difficult to say which of these bones would have united completely and thoroughly. A casual examination of those that show an increased medullary cavity with the attendant thinning of the bony wall, will give one the reason why such bones readily fracture and when fractured why the union is delayed or incomplete.

There can be no doubt of the loss of strength due to the removal of part of the wall from the inside and the additional porosity of the remaining portion. It was thought that these bones fractured because they were more brittle; it seemed that older bones should have less organic material than those at the prime of life, as from ossification *in utero* to the adult period with the completion of the bone growth the percentage of organic material becomes reduced. At birth the compact bones are about 50 per cent organic material, while in the adult the organic material has become reduced to 39 per cent or 40 per cent. It would logically follow that in old age, 60 to 90 years, the organic substance should be farther reduced, but this is not the case as in the older bone the organic material is actually increased to 42 per cent.

The cause of fragility in old bone must be looked for in some other factor. According to the actual condition of increase of organic material, the old bone should be more tenacious and more elastic (properties due to the organic material). In reality the bones are more readily broken. The increase of tenacity and elasticity due to the small increase of organic content is more than balanced by the porosity and the thinning of the bony shell. This can readily be demonstrated in the slides that show the senile changes. Here, although the organic content may be higher, the bone is much weakened by the loss in thickness of the bony wall and the destruction of the canals and systems. When the destructive senile changes are advanced and marked, it stands to reason that such a bone is in no condition to give rise to normal repair activities when fractured. In consequence, although bone repair may be attempted at the fracture, bone destruction is, no doubt, still going on and two conflicting processes are seen. With the age of the patient considered, and his possible lowered vitality, it is no wonder that he becomes bedridden.

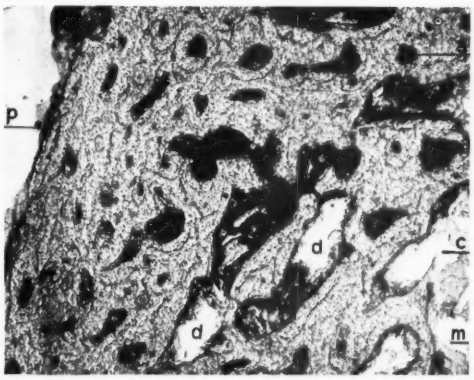

Fig. 8. Ground section of the femur. The normal haversian systems are fairly numerous. *M*, marrow cavity; *P*, periosteal surface; *a,a*, early stages of destruction with individual systems involved; *C*, several large areas of destruction merging with the medullary canal; *d,d*, extensive destruction with the involvement of many systems with the intervening interstitial lamellæ.

While in some elderly persons the physiological process may be very little reduced, in others it is markedly reduced from that of the prime of life; with senile changes in the bones, indicating an altered physiology and histology, it is readily manifest that repair cannot and will not be normal.

The writer has endeavored to give an explanation as to the whys and wherefores of delayed or non-union in the repair of fractures in the elderly and also to give the practitioner a picture of the condition present in such cases. If the healing is much delayed, the prognosis naturally will be unfavorable and the attending physician can clearly explain to the intelligent patient just why no union can be hoped for. While no cure is to be expected, it, nevertheless, places the physician upon a better plane of understanding with his patients.

The writer desires to thank Dr. J. Parsons Schaeffer for his helpful suggestions and criticisms and J. L. Wade for the preparation of the illustrations.

PYELOSCOPY—RADIOSCOPY OF THE KIDNEY PELVIS

A Clinical and Experimental Study[1]

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RADIOGRAPHY of the kidney pelvis has been carried out for a number of years, but only in recent years has the pelvis filled with an opaque fluid been studied with the fluoroscopic screen. As far as the writer has been able to determine the first mention in the literature is by Fey, Truchot, and Dossot (1925), and this present work is inspired by the work of these authors.

ANATOMY

In its lower part the musculature of the ureter consists of an outer circular and an inner longitudinal layer while in its upper part and in the pelvis of the kidney there are three layers, outer longitudinal, middle circular, and inner longitudinal, while the higher one goes more and more strands of fibrous tissue separate the bundles of smooth muscle (Cunningham). The musculature of the wall of the calyx is carried on to and becomes continuous with the muscular tissue in the papilla (Schafer). Embryologically the ureter, pelvis of the kidney, calyces, and collecting tubules in the medulla of the renal pyramids arise from the epithelium of the diverticulum from the junction of the wolffian duct and the posterior segment of the cloaca (Keith). It seems reasonable therefore to expect that these four sections of the urinary duct system should function more or less as a whole. The nerves to the ureter and kidney pelvis are via the renal, spermatic, and hypogastric plexuses (Cunningham) and in the dog have been traced chiefly from the eleventh, twelfth, and thirteenth dorsal nerve roots (Starling). Nerve fibers are known to enter the upper and lower ends of the ureter while throughout its length is a ganglionic nerve plexus (Starling). Engelmann states that contraction waves travel down the ureter at the rate of 20 to 30 millimeters per second.

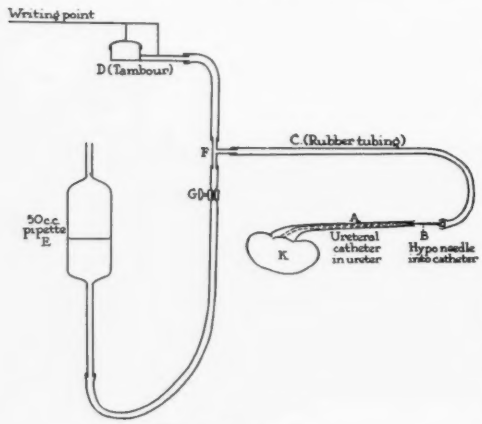

Fig. 1. Apparatus.

METHODS OF THE PRESENT INVESTIGATION

Clinical. A ureteral catheter of about 10 charrière is passed up the ureter in to the pelvis of the kidney and lipiodol or iodipin slowly injected with a syringe until 10 cubic centimeters have been injected or until the patient begins to complain of pain, the injection being generally carried out under the fluoroscopic screen. It is important not to continue to inject after the patient first begins to complain of pain. This point will be dealt with later. The catheter may then be slightly withdrawn, but still left well up the ureter so as to block it. The kidney pelvis may then be observed on the screen, photographs being taken from time to time. All drugs administered were given subcutaneously or intramuscularly.

Experimental. In all cases dogs under chloroform anæsthesia were used and were killed at the end of the experiment while still

[1]From the Department of Physiology of the University of Melbourne, and the Gynæcological and Radiological Departments of the Women's Hospital.

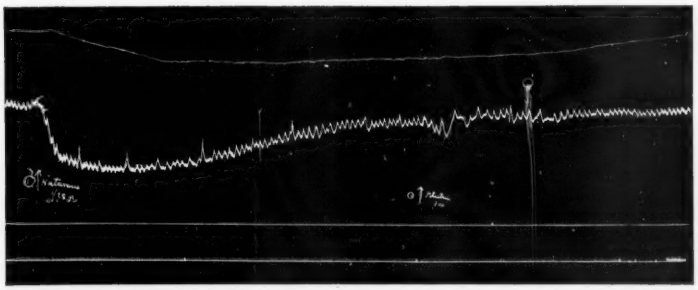

Fig. 2. Histamine.

under the influence of the anæsthetic. In some cases a large dose of morphine (2 to 4 grains) was also given, preliminary to the chloroform anæsthesia (this is our usual procedure here in vivisection experiments).

Pyeloscopy with the fluorescent screen. The technique was identical with that in the human being outlined. All drugs administered were given either intravenously through a cannula in the jugular vein or intramuscularly. This portion of the work was done in association with Dr. H. Flecker and forms the subject of a separate communication later in this paper.

Graphic recording of contraction or relaxation in the renal pelvis. A ureteral catheter is passed up the ureter well into the pelvis of the kidney (its position being verified by subsequent postmortem examination). The method of recording is as follows: The inlying catheter, *A* (Fig. 1), is connected as in the illustration by a large hypodermic needle, *B*, to a length of rubber tubing, *C*, passing to one arm of a T-piece, *F*, a second arm of which is connected with the tambour, *D*, to which is attached a writing point the excursions of which are recorded on a moving smoked paper. The third arm of the T-piece, *F*, is connected to a 50 cubic centimeter or 100 cubic centimeter pipette half-filled with normal saline solution, the fluid level in which is kept at about the same height as the animal's kidney. Urine secreted by the kidney is accommodated in the system, and so the screw-clamp, *G*, is adjusted so that the urine secreted will be accommodated in the pipette, *E*, while any more or less sudden displacement of fluid due to contraction or relaxation of the kidney pelvis will be recorded by the writing point. In the resting condition of the kidney a plateau is thus written by the recording point, whereas if the clamp, *G*, be kept quite closed the writing point gradually and steadily rises and after a while the clamp must be released, otherwise the rubber in the tambour will be stretched so tight that it will not record. This

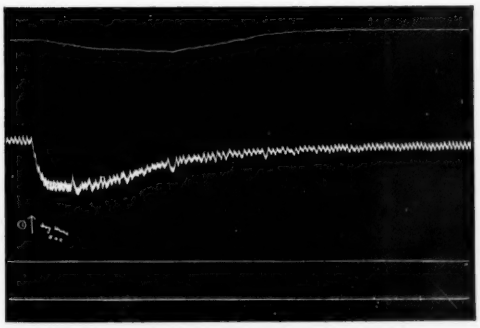

Fig. 3. Dog's urine.

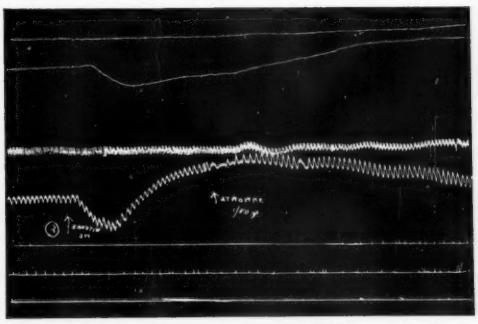

Fig. 4. Ergot.

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RADIOGRAPHY of the kidney pelvis has been carried out for a number of years, but only in recent years has the pelvis filled with an opaque fluid been studied with the fluoroscopic screen. As far as the writer has been able to determine the first mention in the literature is by Fey, Truchot, and Dossot (1925), and this present work is inspired by the work of these authors.

ANATOMY

In its lower part the musculature of the ureter consists of an outer circular and an inner longitudinal layer while in its upper part and in the pelvis of the kidney there are three layers, outer longitudinal, middle circular, and inner longitudinal, while the higher one goes more and more strands of fibrous tissue separate the bundles of smooth muscle (Cunningham). The musculature of the wall of the calyx is carried on to and becomes continuous with the muscular tissue in the papilla (Schafer). Embryologically the ureter, pelvis of the kidney, calyces, and collecting tubules in the medulla of the renal pyramids arise from the epithelium of the diverticulum from the junction of the wolffian duct and the posterior segment of the cloaca (Keith). It seems reasonable therefore to expect that these four sections of the urinary duct system should function more or less as a whole. The nerves to the ureter and kidney pelvis are via the renal, spermatic, and hypogastric plexuses (Cunningham) and in the dog have been traced chiefly from the eleventh, twelfth, and thirteenth dorsal nerve roots (Starling). Nerve fibers are known to enter the upper and lower ends of the ureter while throughout its length is a ganglionic nerve plexus (Starling). Engelmann states that contraction waves travel down the ureter at the rate of 20 to 30 millimeters per second.

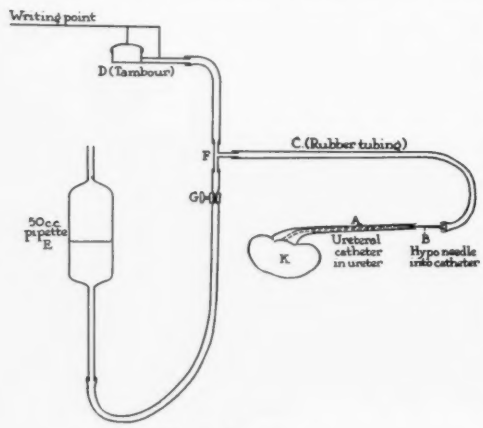


Fig. 1. Apparatus.

METHODS OF THE PRESENT INVESTIGATION

Clinical. A ureteral catheter of about 10 charrière is passed up the ureter in to the pelvis of the kidney and lipiodol or iodipin slowly injected with a syringe until 10 cubic centimeters have been injected or until the patient begins to complain of pain, the injection being generally carried out under the fluoroscopic screen. It is important not to continue to inject after the patient first begins to complain of pain. This point will be dealt with later. The catheter may then be slightly withdrawn, but still left well up the ureter so as to block it. The kidney pelvis may then be observed on the screen, photographs being taken from time to time. All drugs administered were given subcutaneously or intramuscularly.

Experimental. In all cases dogs under chloroform anæsthesia were used and were killed at the end of the experiment while still

[1]From the Department of Physiology of the University of Melbourne, and the Gynæcological and Radiological Departments of the Women's Hospital.

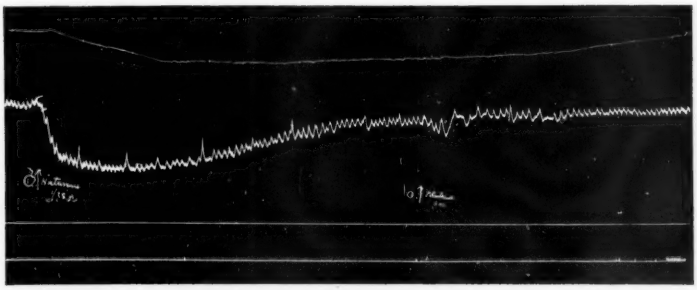


Fig. 2. Histamine.

under the influence of the anæsthetic. In some cases a large dose of morphine (2 to 4 grains) was also given, preliminary to the chloroform anæsthesia (this is our usual procedure here in vivisection experiments).

Pyeloscopy with the fluorescent screen. The technique was identical with that in the human being outlined. All drugs administered were given either intravenously through a cannula in the jugular vein or intramuscularly. This portion of the work was done in association with Dr. H. Flecker and forms the subject of a separate communication later in this paper.

Graphic recording of contraction or relaxation in the renal pelvis. A ureteral catheter is passed up the ureter well into the pelvis of the kidney (its position being verified by subsequent postmortem examination). The method of recording is as follows: The inlying catheter, *A* (Fig. 1), is connected as in the illustration by a large hypodermic needle, *B*, to a length of rubber tubing, *C*, passing to one arm of a T-piece, *F*, a second arm of which is connected with the tambour, *D*, to which is attached a writing point the excursions of which are recorded on a moving smoked paper. The third arm of the T-piece, *F*, is connected to a 50 cubic centimeter or 100 cubic centimeter pipette half-filled with normal saline solution, the fluid level in which is kept at about the same height as the animal's kidney. Urine secreted by the kidney is accommodated in the system, and so the screw-clamp, *G*, is adjusted so that the urine secreted will be accommodated in the pipette, *E*, while any more or less sudden displacement of fluid due to contraction or relaxation of the kidney pelvis will be recorded by the writing point. In the resting condition of the kidney a plateau is thus written by the recording point, whereas if the clamp, *G*, be kept quite closed the writing point gradually and steadily rises and after a while the clamp must be released, otherwise the rubber in the tambour will be stretched so tight that it will not record. This

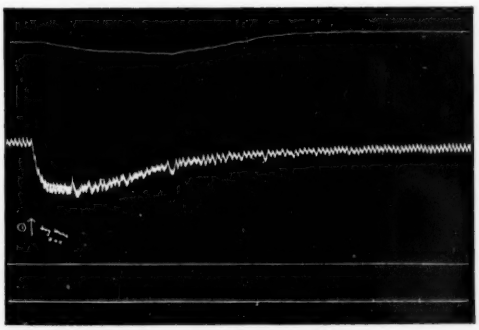


Fig. 3. Dog's urine.

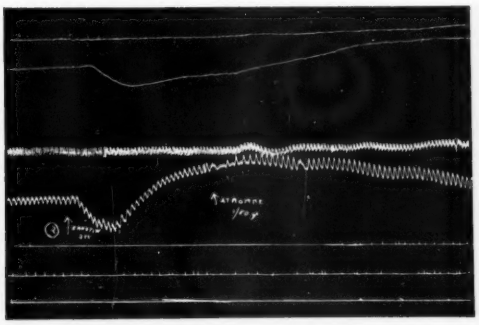


Fig. 4. Ergot.

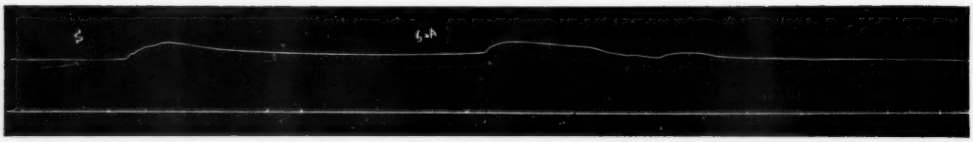


Fig. 5. Strychnine and strychnine and atropine.

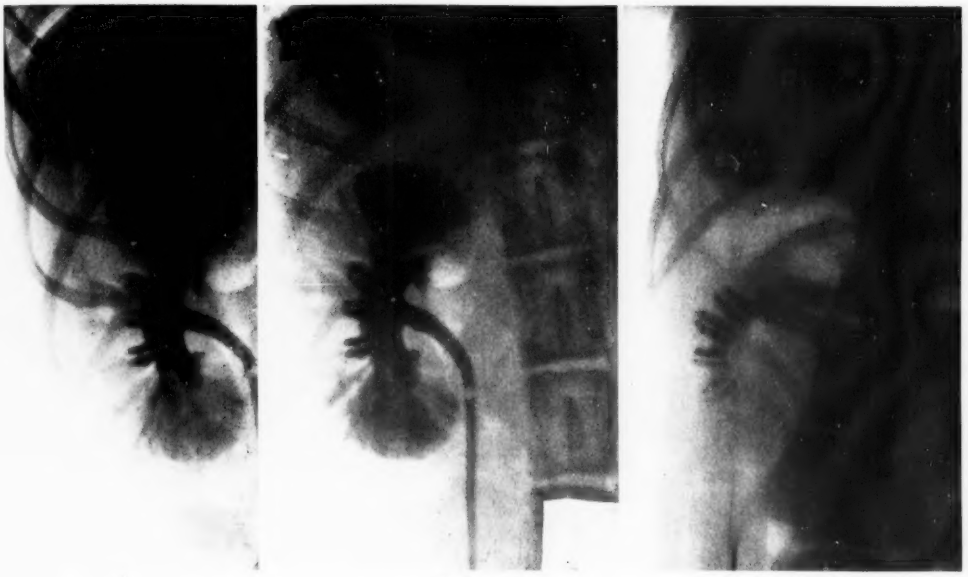
apparatus was suggested by the apparatus which is used by Professor W. A. Osborne, of Melbourne, for recording venous pressure. A catheter which was passed into the pelvis of the other kidney was connected with a drop recorder and indicated the rate of urinary secretion.

Results. As indicated in a preliminary note published by the author (1928), the musculature of the kidney pelvis can be seen to exhibit regular rhythmic contractions. As far as the author has been able to observe the wave of contraction commences in the upper calyx followed in succession by the middle and lower calyces at intervals of from 1 to 3 seconds. During this period the body of the pelvis (for which the name "ventricle of the pelvis" was suggested) undergoes steady relaxation. When this part of the pelvis has received the contents of the calyces the communication channels between the calyces and the "ventricle" become contracted, the "ventricle" contracts with a snap-like action reminiscent of the action of the cardiac ventricle, a "globule" appears at the upper end of the ureter and is passed down the ureter by a peristaltic wave, sometimes so fast as to be barely visible and sometimes so slow that an individual globule can be readily followed in its course down the ureter (when the inlying catheter has been previously removed, of course). Various modifications of this normal functioning have been observed—for example, in one case in which the patient had complained of vague backache it was seen that when the "ventricle" contracted some of the fluid was forced back through the communication channel to the upper calyx which became forcibly dilated and this movement synchronized with the "throbbing" pain which the patient described. In this patient it was observed that an injection of eserine sulphate (1/100 grain) restored the function to normal and at once relieved the pain. In another case antiperistaltic waves up the ureter carried the lipiodol up the ureter into the kidney pelvis. An injection of morphine sulphate "cured" this condition.

Action of drugs. Atropine invariably caused relaxation of the kidney pelvis with overfilling and pain in the human subject. In the dog in some cases a preliminary slight contraction was observed. Morphine, strychnine, eserine, pituitrin, all caused contraction of the kidney pelvis with instant relief of the pain caused by atropine. In the case of pituitrin, the contractions of the ureter were also accelerated to such a degree that it was impossible to see the "globule" passed down it after the "ventricle" contracted. In the experimental animal it was also found that these drugs exerted a like action although contraction was not always readily elicited when the animal was under the influence of a large dose of morphine. The marked relaxing effect of histamine (ergamine and dog's urine) (Fig. 2) which also produced a marked vasodilatation with fall in blood pressure is well seen in the tracings illustrated (Figs. 2 and 3). In small doses histamine produced slight contraction of the kidney musculature. Ergot (ernutin) produced a preliminary relaxation followed by a prolonged contraction (Fig. 4). Strychnine produced a marked contraction while a mixture of strychnine and atropine produced a contraction followed by some degree of relaxation (Fig. 5).

RESULTS AND CONCLUSIONS

1. Pyeloscopy gives us a ready method of determining the pathology of cases of kidney pain where lithiasis is absent. In many cases of pyelitis the author has determined by direct observation which drug gives the best contraction of the renal pelvis and the patient is kept on this for some time. As a general rule,



A. B. C.

Fig. 6. A, Commencing subcapsular bursting of kidney; B, further stage; C, control, reversed in printing. Experiment June 5, 1929.

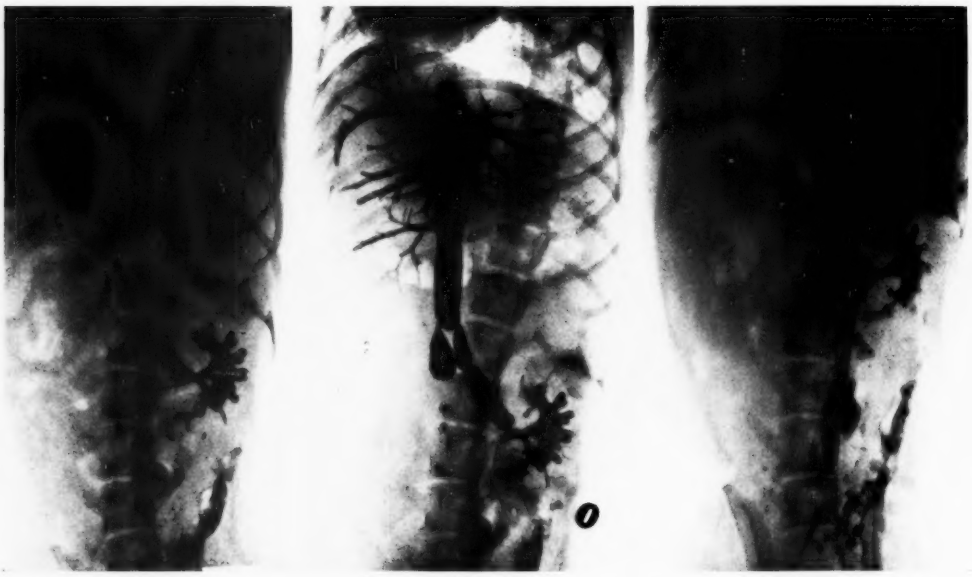
eserine and morphine have been found the best. Patients who had wearily dragged on for years suffering from backache with periodic exacerbations of headache, sweating, rise in body temperature, etc., have derived immediate relief from strychnine, eserine, and morphine, either alone or combined, when citrates and the usually recognized treatments have failed.

2. The cases of antiperistalsis explain how the pelvis of the kidney may be infected from an infected bladder. The pain accompanying these antiperistaltic contractions or the antidromic passage of urine suggest this as a possible cause of the cases of renal and ureteral colic simulating stone when no stone is demonstrable.

3. In cases of hydronephrosis in which the pelvis contracts well and vigorously, obviously the treatment is plication, whereas if the pelvis does not contract and is simply an inert atonic sac, then nephrectomy is obviously indicated.

4. In cases of early malignancy involving perhaps only a single calyx, the absence of contraction of one part of the pelvis associated with "idiopathic" hæmaturia of renal origin would justify the performing of an exploratory operation.

5. The actions of the various drugs illustrated remind us that these drugs commonly administered for various specific conditions have an action and produce effects on other organs of the body, and the effect on the kidney musculature might produce unexpected symptoms. Histamine, which has come into such prominence in recent years more especially in regard to surgical shock has a very definite physiological action on the kidney musculature. A whole vista of possibilities is opened up—what effect this action would have on the elimination of urine from the kidney itself, what influence it might have in the causation of stagnation of urine and back-working into the kidney collecting tubules, how much of the symptoms of pyelitis are due to the local elaboration of histamine-like bodies by the action of the bacteria which have invaded the kidney pelvis, what is the influence of colonic stasis with the absorption



A. B. C.

Fig. 7. A, Commencing injection 10 cubic centimeters of solution at 4:56 p.m.; B, after injection at 5:02 p.m.: C, control experiment May 29, 1929.

of sympathomimetic amines on the excretion of urine, and many other of the problems associated with the therapeutics of kidney conditions in general.

6. Too much stress cannot be laid on the necessity for slowly injecting the kidney pelvis and the great danger of a rupture incurred if nature's warning of pain be ignored. (In the following paragraphs is appended a brief description of the work which has been carried out by the author in collaboration with Dr. H. Flecker.)

Experimental Pyeloscopy in the Dog[1]

Observations with the fluoroscopic screen of the pelvis of the dog's kidney filled with an opaque fluid (lipiodol or sodium iodide solution) showed the same responses to drug action as the human, viz.:—relaxation with atropine and a contraction with morphine, strychnine, eserine, pituitrin, ergot, and pilocarpine.

[1]From the Departments of Physiology and Anatomy in the University of Melbourne.

A point of very great practical importance elicited in these experiments was the ease with which the kidney pelvis could be ruptured. In one case a head of pressure of about 35 centimeters potassium iodide solution (30 per cent) caused a rupture. As seen in the radiograms (Fig. 7) the rupture is generally subcapsular and is usually at the lower (caudal) end of the pelvis. Very soon, however, the opaque fluid enters the veins and in one picture the fluid is seen in the intercostal veins (Fig. 7). This is obviously of very great importance in the technique of filling the renal pelvis with a fluid under pressure, as when a pyelogram is to be taken in the human subject, and apparently accounts for fatal cases in which a non-viscid fluid such as sodium iodide solution has been rapidly injected under pressure. Apparently the pain felt by patients when the pelvis is reaching "breaking point" is nature's warning signal and must on no account be disregarded. This question is being further investigated and it is hoped will be the subject of a further communication later.

Details of some experiments, relative to rupture of the kidney

Dog No. 34, F., tan terrier. On May 29, 1929, after a preliminary injection of 2 grains of morphine a cannula was inserted in the left ureter and 4.5 cubic centimeters of "ioran" (iodized oil) run in. Later 10 cubic centimeters was injected. Necropsy showed rupture of lower calyx of pelvis, apparently into the renal vein. The renal vein and vena cava contained drops of oily liquid.

Dog No. 31. On May 15, 1929, 5 cubic centimeters of ioran was injected into each kidney through cannulas in each ureter, and then a further 10 cubic centimeters was injected into the left ureter. The X-ray picture showed a great bulge in the lower part of the kidney as if the kidney were ruptured.

At necropsy the left kidney showed a small rupture in the lower pole of the kidney from the lower calyx under the capsule. The capsule was distended with ioran on the lower and mesial (anterior) aspects of the kidney. The right kidney showed a small ecchymotic patch about 0.5 centimeter in diameter.

On May 6, 1929, potassium iodide solution (30 per cent) was run in at a head of pressure of 1 meter of the solution. In 30 seconds the pelvis was filled. In 4 minutes the kidney was found burst.

My thanks are due to Professor W. A. Osborne for the use of the laboratories of the Department of Physiology of the University of Melbourne and for his advice while this work has been in progress, to the medical superintendent of the Women's Hospital, Melbourne, for placing the facilities of the hospital at my disposal, and to Sister Blyth of the radiological department of the Women's Hospital, Melbourne, for her technical assistance in the clinical investigation.

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LIPIODOL PELVIC CYSTS[1]

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SINCE the beginning of the use of iodized oils as opaque media for roentgen examination of the uterine and tubal cavities, there has been present the question of the effect of these oils on the mucous membrane of these cavities and on the peritoneum when spill of the oil from the tubes occurred. Sicard and Forestier, from a large experience with roentgenological exploration of spinal and cerebral spaces, genito-urinary organs, and other organ cavities, considered the injection of lipiodol non-irritating to the various cavity linings. They stated that lipiodol was a definite chemical compound, with 40 per cent iodine bound to poppy seed oil, and therefore, not a solution and tolerated by human tissues and cavities as any ordinary oil would be. They also stated that very high doses gave hardly any reaction and generally no iodism, as no free iodine was present. However, they cautioned against the use of samples of brownish color or dim appearance which indicated free iodine.

Following this reassuring study, other reports appeared. Stein and Arens, Stone and Jarcho (the latter gives a long list of references to other workers) stated that lipiodol was harmless in their experience with large series of patients.

The therapeutic use of iodized oils was then introduced in gynecology after having been used in pericardial, pulmonary, and other disease conditions. Cotte and Bertrand, in 1926, observed that lipiodol had a powerful antiseptic action and would destroy any micro-organisms in the fallopian tube after intrauterine injection. Although they used lipiodol for diagnostic purposes in a large number of cases of acute or subacute inflammation of the fallopian tubes, they never noted any rise in temperature or any unfavorable reaction due to the introduction of the oil. On the basis of these observations they suggested the therapeutic use of lipiodol early in the course of salpingitis, assuming that it might result in early recovery and prevent obstruction.

[1]Read before the Chicago Gynecological Society, November 15, 1929.

Another favorable report was made by Cotte and Pierre, who gave intra-uterine lipiodol injections in 20 cases of acute adnexal inflammation, without harm. The iodized oil was thought to have a favorable influence on the pathological process in the fallopian tube. In these reports, there was a disregard for dosage of the oils and infection of the genital tract.

In contrast to such overwhelming favorable results demonstrating freedom from danger in the use of the iodized oils, only few reported unfavorable results were reported. Odenthal had two patients who developed pathological changes from the injection of the contrast material. In one patient he injected only 4 cubic centimeters of 40 per cent iodipin. Within 10 days after the injection, a bilateral tubal infection began which subsided under conservative treatment after 3 weeks. He operated on the second patient 3 weeks after the injection. Microscopic sections of the tubes showed oil and giant cells in the wall. He thought that these cases illustrated the dangers of the use of iodized oils in spite of asepsis and indications.

Lecène and Béclère report the first accidental infection of the fallopian tubes in a series of 230 observations. Descomps, Delassus, Sicard, and Solal, each had a patient who developed a pelvic infection after lipiodol injection. Descomps found lipiodol in the cul-de-sac of another patient, at operation, 2 months after injection.

Ries published in the transaction of the Chicago Gynecological Society a very thorough report of a case of a woman, who was operated upon 6 months after the injection of lipiodol. He found extensive adhesions between the peritoneum, omentum, uterus, bladder, sigmoid, and appendix. After freeing the omentum he found cysts and walled off fluid among the thin adhesions. The fluid was sterile and gave no iodine reaction. On the under surface of the mesentery of the small intestine there were 25 stone-like formations embedded in the serosa of the mesentery. No iodine was found in these masses which had a cheese-like structure held together by some strands of connective tissue. These masses were found in the adhesions of the pelvis also. In the microscopic sections of the tubes, large numbers of giant cells were seen, in which were homogeneous greenish masses and granular masses.

A recent experimental study of the effect of iodized oil on serous membranes was published by Crandall and Walsh. They found that lipiodol and lipoiodin were irritating to the lining of the pericardial, pleural, and joint cavities while the oils appeared harmless in the peritoneal cavity.

As there are only 2 case reports in the literature of pathological changes in the pelvic tissues (Odenthal, Ries) and only one of the presence of lipiodol 6 months after the injection of the oil (Ries), the following case of the patient with a uterus bicornus unicollis, with lipiodol in the pelvis 22 months after injection during which period she delivered a full term child, is unique.

F. G., a colored woman, 23 years old, entered the obstetrical department of Cook County Hospital on September 21, 1929. Her chief complaints were pain in the lower abdomen, of 4 years' duration, but aggravated in the last 2 months, and a yellowish white vaginal discharge of 5 years' duration. Her description of her complaints was that of chronic adnexal disease. Menstruation occurred irregularly, at 4 to 6 weeks' intervals, for the last 6 months, lasting 7 to 10 days. The flow was profuse and was associated with lower abdominal pain. In 1925, she delivered a premature child (7 months).

On August 18, 1927, although only 8 months' pregnant, she had brought on labor by taking castor oil for constipation. She was seen by me for the first time after having been in labor for 13 hours, with the bag of water ruptured for 8½ hours. Examination showed a normal full term baby in the right occipito-posterior position, head high, due to an irreducible obstructing mass, which was being further impacted into the left side of the pelvis by the presenting part. A low transverse cæsarean section was performed, complicated by a short longitudinal laceration upward in extracting the head manually. The obstructing mass was found to be the left horn of a bicornuate uterus flexed laterally and forced into the pelvis by the head. At this time, gonococci and streptococci were found in the vagina. The Wassermann reaction was positive. She developed a puerperal fever (acute metritis, septicæmia), which responded favorably to therapy which included puerperal fever streptococcus antitoxin. The premature labors may be attributed to the uterine anomaly rather than to the lues because of the normal appearing placenta. This characteristic of bicornuate uterus has been described and emphasized by Falls.

The patient returned in October, 1927, and a late secondary luetic skin eruption with cervical lesions

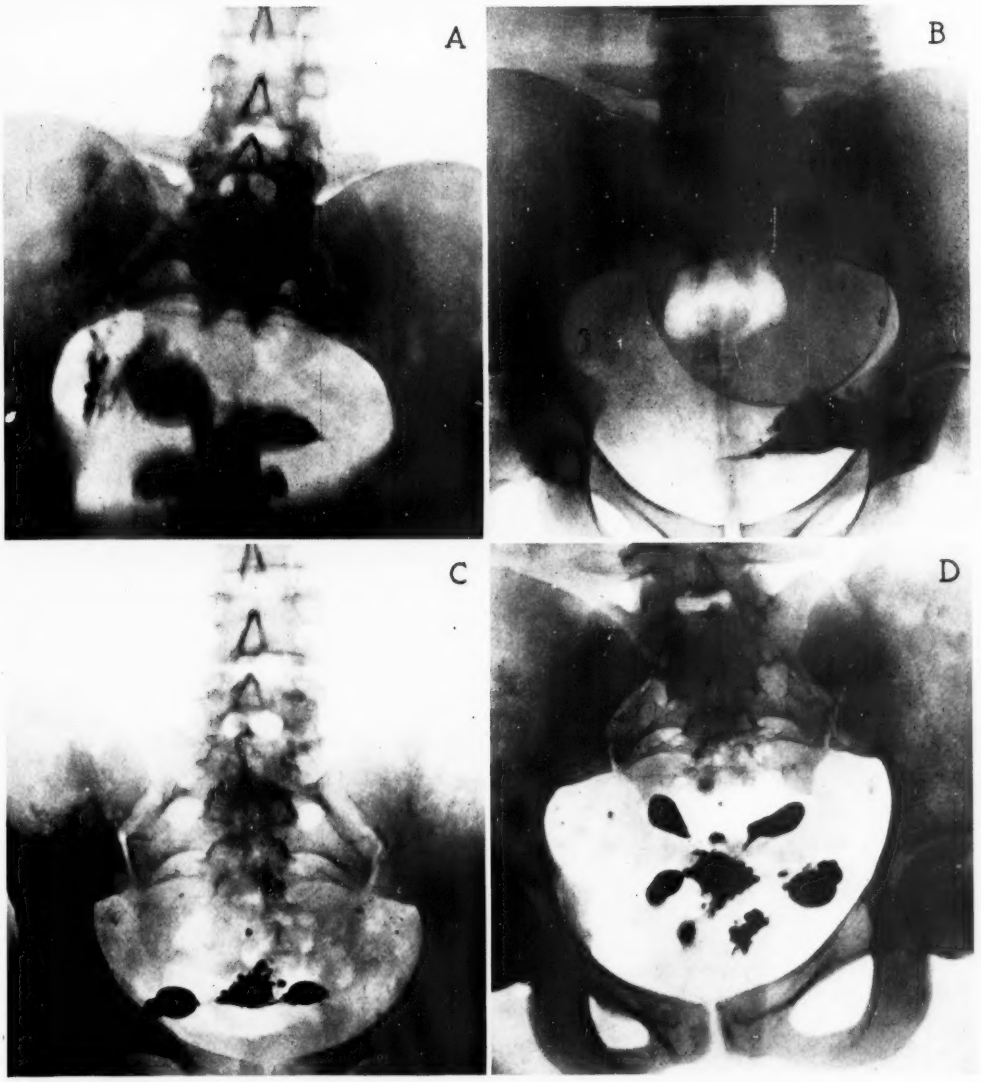

Fig. 1. Roentgen plates of *A*, pelvic viscera after lipiodol injection, December 27, 1927; *B*, fetal head in pelvis, lipiodol in pelvic cavity, October 28, 1929; *C*, pelvis and lipiodol cysts, September 14, 1929; and *D*, pelvic viscera after lipiodol injection, September 14, 1929.

was found. She was given a course of antiluetic treatment. The cervical discharge subsided and smears did not show any gonococci on December 3, 1927. Lipiodol (6 cubic centimeters) was injected into the uterus and a roentgen plate was made but proved unsatisfactory. Therefore, on December 7, 1927, 10 cubic centimeters of lipiodol was injected and roentgen examination showed the two uterine cavities, larger than normal, patent tubes, and some of the oil in the pelvic cavity. Following this examination the patient was observed for weeks and no apparent complication developed.

In June, 1928, the patient came back again because of pain in the lower abdomen. Bimanual

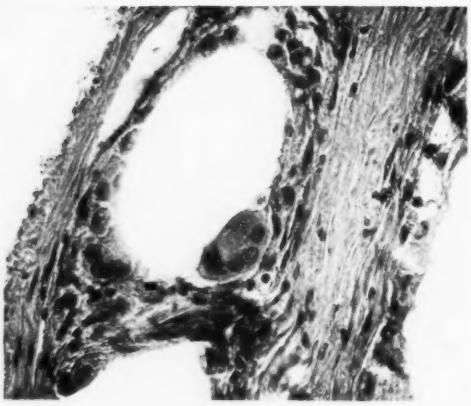

Fig. 2. Photomicrograph of cyst wall illustrating histocytes and giant cells. × 325.

examination found the left horn pregnant. Roentgen examination showed that lipiodol was present in the pelvic cavity. In October, 1928, the roentgen plate showed a well developed fetus to the left and lipiodol in the pelvic cavity. On December 26, 1928, patient began to experience labor pains and she went to the door to call for a taxicab when she had one strong pain, fell on the floor, and a full term normal baby was born precipitately. As she stayed at home the details of the puerperium were unknown. Pain in the lower abdomen as described forced her to return for local treatment.

As to her past history she has had measles, malaria, and typhoid fever. There was nothing of significance in her family history. All three pregnancies were illegitimate.

Physical examination November 21, 1929, revealed a well developed, well nourished colored female about 25 years of age having a normal temperature, pulse, and respiration. The essential findings were: the abdomen was round, firm, and elastic with a subumbilical midline keloid scar; no masses or rigidity were present but definite pain was produced by pressure in the iliac fossæ. On vaginal examination the vulva was found normal, the pelvic floor was firm and elastic, the cervix was hard and round, but its regularity was interrupted by an anterior septum 4 to 5 millimeters thick extending along the anterior surface of the portio and anterior vaginal wall. The external os was patent with a depression posteriorly. The corpus was represented by two firm cylindrical masses about 5 to 6 centimeters long, and 3 to 4 centimeters in diameter, lying high in the pelvic cavity being fixed and tender on motion. The adnexa bilaterally were fixed and tender but no masses could be defined. Speculum examination showed the cervix with a simple erosion and the anterior vaginal septum. Urinalysis revealed no pathological findings. Wassermann was negative. The diagnosis was made of bilateral chronic salpingitis with healed pelvic peritonitis with retained masses of lipiodol. On September 14, 1929, roentgen examination of the pelvis showed lipiodol still present. Lipiodol was injected again, demonstrating the two uterine cavities with closure of the tubes at their uterine ends in addition to the old lipiodol masses.

A laparotomy was performed on September 23, 1929. When the abdomen was opened, the omentum was found firmly adherent to the parietal peritoneum anteriorly near the lower end of the wound, to the right uterine body and adnexa, to several cystic masses in the anterior cul-de-sac, to the point of fusion of the two horns of the uterus, and to the top of the bladder. The adhesions were firm, and in order to free the omentum sharp dissection was necessary. The three cystic masses found in the interstices of the adherent omentum in the anterior cul-de-sac varied in size from about 1.5 centimeters to 3 centimeters in diameter. The two smaller ones contained a clear, light amber, oily fluid, while the larger one contained a dark brown oily fluid with yellow flakes which came from the lining of the cyst wall which was about 1 millimeter thick.

The bicornuate uterus was high in the pelvic cavity, lying just above the sacral promontory. Both tubes were thickened, firm, tortuous, and bound down with firm adhesions to their respective ovaries. The fimbriated end of the right tube was open. The right ovary was larger than normal, made up of multiple small cysts and one hæmorrhagic cyst. A supracervical hysterectomy, bilateral salpingectomy, and right oophorectomy were done. The convalescence was uneventful except for an acute pyelitis and wound hæmatoma. The patient left the hospital in good condition. Microscopic sections of each corpus uteri showed an œdematous resting endometrium, a thickened myometrium with areas of round cell infiltration which were also present in the serosal adhesions. Sections of the ovary showed multiple follicular cysts and one corpus luteum. The sections of the isthmic and ampullar portion of the right fallopian tube showed subepithelial and perivascular round cell and plasma cell infiltration. The section through the lipiodol cyst showed a thin connective tissue capsule lined with histocytes, and outside of the capsule two foreign giant cells were seen in a smaller cyst.

The oil in the cysts was sterile and gave no iodine reaction. However, on breaking the lipiodol down (according to the instructions given in the *New and Non-official Remedies* 1928, page 214, for iodipin), a positive reaction for iodine occurred. The yellowish white flakes found in the oil and on the surface of the cysts were amorphous under the microscope.

SUMMARY AND DISCUSSION

From an analysis of the history and clinical course of the conditions and pathology in this patient, one is lead to separate the various entities. There was a chronic bilateral salpingi-

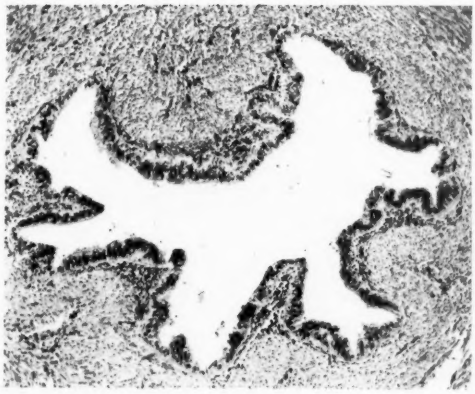

Fig. 3. Photomicrograph of fallopian tube illustrating subepithelial round cell infiltration. × 85.

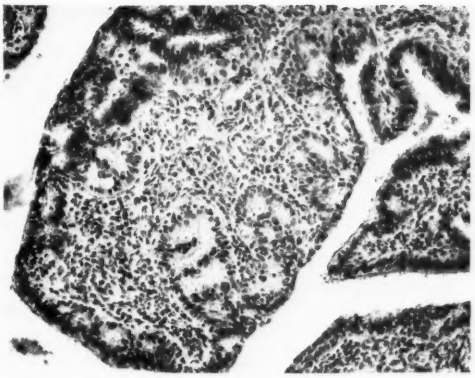

Fig. 4. Photomicrograph of the endosalpinx illustrating chronic inflammation. × 325.

tis with healed pelvic peritonitis in which gonococci and streptococci probably played a rôle. Also the lipiodol had produced a foreign body reaction in the pelvis with the formation of cysts and giant cells independent probably of the pelvic infection. The intimate association of the omentum and the lipiodol may indicate that it plays an important part in the removal of the lipiodol from the pelvis. Schochet mentioned this function of the omentum in discussing Ries' report. After the lipiodol injection, the tubes were functionally competent since pregnancy occurred one year later. With repeated infections as might be expected in this type of individual, the tubes were finally closed at the uterine ends (salpingitis isthmica nodosa), as demonstrated by the last roentgen plate. The lipiodol was sterile and gave a positive reaction for iodine only after it was saponified, thus indicating chemical combination rather than solution of iodine in poppy seed oil. In considering the large number of favorable reports with absence of complications after the injection of iodized oils into the uterus as compared with the very few reports of complications one is impressed with the harmlessness of these oils in the pelvic structures. The absence of follow-up in many cases, however, may explain this apparent infrequency of complications although the iodized oils are so generally used.

That the lipiodol is antiseptic is questionable because the iodine is in chemical combination and therefore not free to act and because the oil does not mix with liquid media. In order to test the antiseptic value of lipiodol I mixed equal parts of lipiodol and a 24 hour broth culture of streptococci in one tube, and of colon bacilli in another tube. Control tubes of equal amounts of broth culture of these organisms were used. The oil culture mixtures were shaken for 3 to 5 minutes and all the tubes were incubated for 24 hours at 37.5 degrees C. After the mixtures were again shaken, subcultures were made on blood agar plates which were incubated for another 24 hours. The number of colonies on the plates from the controls and the lipiodol culture mixtures was the same. It was evident that the oil and the broth did not mix but the shaking would be expected to bring small particles of the oil in contact with the bacteria. The lipiodol did not inhibit growth although bacteria did not grow in it as it remained clear. Also Dr. I. Pilot has informed me that he has used lipiodol to help produce lung infections by keeping the introduced bacteria in the bronchioles. Therefore, the antiseptic properties of lipiodol are still to be proved. In intrauterine injections the oil may well force or carry along with it infected secretions from the cervix or tubes into the pelvic cavity to produce an infection which varies in its severity. The acute types become apparent early, while low grade infections over a long period of time may be symptomless.

The cases of Odenthal, Ries, and the one here described would suggest a foreign body reaction in the pelvis to lipiodol. I believe that there are two possible explanations for this reaction. First, there may be sufficient free iodine to be irritating and yet not grossly visible or the oil acts as a foreign body in some patients. Second, in the presence of old pelvic adhesions, the peritoneal surfaces may be so changed that there is a decreased vascularity. The response of the histocytes or macrophages may be insufficient to cope with the amount of lipiodol to be phagocytized. Therefore, the fibroblasts form around these masses with the surrounding macrophages and wall off the foreign body. The omentum takes part in this walling off process. The possibility of producing a foreign body reaction by the lipiodol cannot be utilized as an argument against its use since this complication is rare in comparison with the frequency with which lipiodol is used. The danger lies only in the use of the chemically changed sample of the oils or in the presence of infection of the genital tract.

Although further studies are necessary to settle the problems discussed, the following conclusions may be made from an analysis of previous case reports and the present case.

1. In a case of uterus bicornis unicollis bilateral chronic salpingitis and healed pelvic peritonitis occurred independently of the foreign body reaction produced by the presence of lipiodol in the pelvic cavity. The function of the tubes was not disturbed by the lipiodol as the patient delivered a full term child one year after injection.

2. In the presence of infections of the female generative tract, the use of iodized oils is contra-indicated as their antiseptic properties have not been demonstrated.

3. In some individuals lipiodol may induce foreign body reaction in the pelvis but it is also true that some specimens of lipiodol (free iodine) may be irritating to any peritoneal surface.

4. The great value of iodized oils in roentgen examination of the female pelvic organs should be appreciated but due precautions in its use must be observed. It is suggested that the oils be tested for free iodine and that oil injections be used only in the absence of infections of the female generative tract.

I wish to acknowledge the great help rendered by Miss Louise Montgomery of the Social Service Department of Cook County Hospital in following this patient.

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URETHRAL CARUNCLE IN THE FEMALE

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IN routine pathological diagnosis, some cases of relatively minor importance seem more perplexing than frankly malignant conditions. One of these border-line conditions is the so-called "caruncle" of the female urethra. In this location, the infolding of the epithelium is marked enough to raise the question of carcinoma, although no carcinoma exists. Before considering the pathological changes, a review of the development and normal histology of the urethra is necessary.

EMBRYOLOGY OF URETHRA

By the fourth week of intra-uterine life, the cloaca is formed from the entoderm near the posterior pole of the embryo, and this gives origin to the allantois (Keith). By the seventh week the entodermal cloaca is represented practically exclusively by the urogenital sinus. Later, separate openings develop in this to form, in the female, the vagina and urethra. According to Wood-Jones the female urethra represents the cloacal remnant in its simplest form, but others believe that the trigone of the bladder also arises from the cloaca. In either case, the bulk of the bladder develops from the allantois.

ANATOMY AND HISTOLOGY

The female urethra is composed of three layers: the mucosa, submucosa, and a muscular coat. In the mucosa are numerous small glands. The submucosa contains cavernous tissue, and near the meatus the two paraurethral glands of Skene. The muscular coat has an outer circular layer of smooth muscle, which is thickest at the bladder, and an inner longitudinal layer. Veins are present between these (Morris). At the vestibular orifice is the external sphincter, formed of striated muscle (Schaefer).

"The epithelium itself," says Kaufmann, "shows marked individual variations, being composed of layers of squamous epithelium (lower part), or of transitional epithelium (above)." Bailey refers to it as stratified squamous epithelium of transitional type near the bladder, then two-layered stratified, or pseudostratified; and, exteriorly, stratified squamous epithelium. According to Lewis and Stoehr, the epithelium has been variously described as stratified, with outer squamous cells, or as pseudostratified and columnar. It may be of different forms in different individuals. The lumen is irregularly crescentic with longitudinal folds.

We have studied transverse and longitudinal sections of several normal female urethras. In these, the epithelium of the inner end seems to be indistinguishable from the transitional epithelium of the bladder. That of the meatus has shown both fairly typical squamous and transitional epithelium, sometimes both in the same transverse section. We have found no evidence of hornification to differentiate the two types clearly, but the scale-like shape of the cells of the superficial layers of the squamous epithelium is quite unlike the polygonal cells of the transitional type.

PATHOLOGY

The urethral caruncle or vascular tumor of the meatus was first described by Samuel Sharp in 1750. It is a growth of the lower half of the external orifice, florid or dusky red. It is pedunculated, or on a broad base, says Kelly, who describes the papillæ, the dilated vessels, and the pavement epithelium. He says that the presence of an unusual number or arrangement of nerve fibers has not been demonstrated. Schroeder, in a recent publication refers to the dilatation of the vessels, the plasma cell infiltration, and the fact that, in all of his 6 cases, the epithelial thickening is of stratified squamous epithelium. The vascular element is also emphasized by Conrad, Ferrier, Howze, and Schmitt (12, 13); while Young seems especially insistent on the chronic inflammatory changes. Kaufmann says: "The so-called 'urethral caruncles' which appear in women close to the orifice of the urethra and project from the surrounding

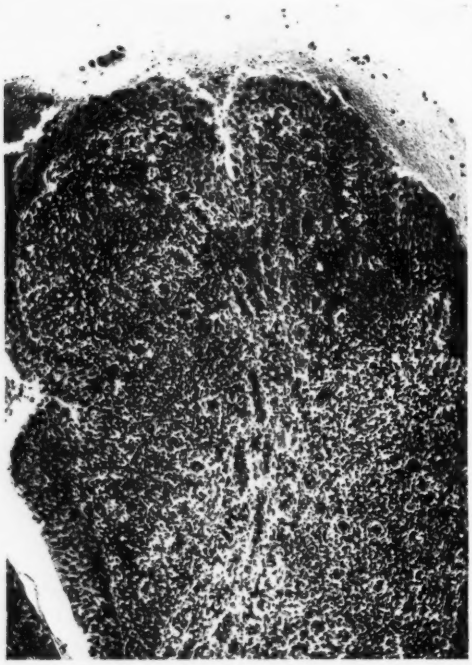

Fig. 1. Case 1. A characteristic picture of urethral caruncle showing transitional epithelium, chronic inflammation, and numerous vessels (× 40).

surface, are outgrowths of mucous membrane; they take on the character of granulomata, and partly of hypervascular, papillary, or telangiectatic mucoid polyps; they may reach the thickness of a small finger and a length of several centimeters and prolapse from the urethral orifice. They often contain adenoid tissue and glands, possibly cystic."

Ewing says: "A common error in the diagnosis of urethral carcinomata in the female results from the deep normal invaginations of the epithelial lining of this structure. On section, these invaginations may appear as displaced islands of atypical epithelium, strongly resembling epithelioma."

In the past few years we know of two cases in which extensive operations have been performed for benign urethral caruncle because of erroneous diagnoses of epithelioma. The records of the New York Hospital do not show a single case recorded as carcinoma of the female urethra.

The etiology of caruncles, as described in the literature, seems uncertain or varied. Childbirth, apparently chiefly through its relation to infection, seems to predispose, as does also gonorrhœa. The most likely age of origin is described as at or about the menopause. The chief complaints are usually marked dysuria and frequency.

CASES STUDIED

Of the 23 specimens we have studied, 3 will be referred to separately, the others only in a tabulation of the entire series.

Case 1. No. 12,068, a patient of Dr. L. L. Fulkerson's, Cornell Clinic, was a married woman of 60 (in 1927) who has had several children, the last 18 years previously. Venereal history was negative. She has been treated at the general medical clinic for constipation, etc., since 1922, but the first observation relative to the bladder or urethra was made in October, 1925. At that time there were symptoms of frequency and pain on urination, and a diagnosis of "prolapse of urethra" was made by one of Dr. Fulkerson's predecessors. Silver nitrate was applied twenty-one times in the next 16 months, without relief of symptoms. When first seen by Dr. Fulkerson, March, 1927, he made the diagnosis of "urethral papilloma," of the left posterior margin of the urethra, and the specimen was regarded by him as possibly malignant. The laboratory found chronic inflammatory changes only, with a picture similar in all respects to that shown in Figure 1. Two weeks later, the papillomatous growth was completely removed and the base cauterized. There were several fairly large papillary projections.

The tissue received in the laboratory consisted of two soft papillary masses of light pink tissue, each about 2 millimeters in all diameters. Microscopically (Fig. 1), the epithelium of the specimen was transitional in type, with rarely over three or four cells in its thickness, and was infiltrated with polynuclears. The stroma showed much round cell infiltration, and, in the numerous small vessels, there were many polynuclears. There was a small, branching, gland-like mass in the submucosa. This was lined with a single row of columnar cells, and below this there were several layers of ovoid cells. This was considered a portion of a para-urethral gland of Skene. In summary, this specimen in the main showed little but the usual inflammatory reaction. However, there was one area in the section (Fig. 2), which presented a different picture. Here, the epithelium was markedly infolded and thickened, forming a large nidus. The cells of this mass showed little or no hyperchromatism and were fairly uniform in type. They were of a fairly uniform size and showed normal orientation. No mitoses were found. There were many round cells in the adjacent stroma. Owing to the infolding, the question of possible malignancy arose, but the

Fig. 2. Case 1. Showing "infolding" of epithelium (× 40) in a benign urethral caruncle.

Fig. 3. Urethral caruncle with gland of Skene in the submucosa (× 38).

case was referred to Dr. Ewing, who diagnosed it as entirely benign.

When last seen, 8 months after the operation, the case was pronounced "cured." There was no evidence of recurrence, and all symptoms had disappeared.

CASE 2. No. T113, 1927. A woman of 55 years was purported to have had removed 6 years previously a caruncle of the same type as that of the present growth. Dr. W. S. Hastings sent the slide to Dr. Ewing, who confirmed Dr. Hastings' findings of a benign process. The epithelium was thickened, transitional in type, and the picture was similar to that in Figure 2. The course was subsequently uneventful with no symptoms or signs 8 months later.

CASE 3. No. T173, 1927. A woman of 63 years of age had a caruncle of the urethra. In this case also a slide was sent to Dr. Ewing by Dr. Hastings and again the epithelium was found to be of the transitional and infolded type, and the picture resembled that shown in Figure 2. A good deal of acute inflammation was present in the stroma, and there was one small acinar gland lined with columnar cells. Some of this lining layer of cells contained drops of mucus. The diagnosis was caruncle. The subsequent course was uneventful, with no signs or symptoms 7 months later.

SUMMARY OF CASES—CLINICAL FACTORS

We have collected 23 cases of caruncle which number includes 19 cases from the New York Hospital, in which the slides were satisfactory and the history fairly complete. The 3 cases of which brief summaries are given in this article are included in the tabulation.

The ages of the 21 women varied from 23 to 55 years; the average age being 51 years.

In the 15 cases in which information regarding the marital status was available, we find 10 married with children, 3 married without children; and 2 unmarried women.

No history of venereal infection was noted.

The duration of symptoms of burning and dysuria was noted in 18 cases. In one case each, this had lasted 16, 14, 12, 7, and 6 years. Most of the remaining cases complained of symptoms for a period of about 1 year. The shortest history was 1½ months.

Complications which were noted are: six instances of relaxation of the pelvic floor with

several additional cases in which this condition seemed probable; two of hæmorrhoids, (one developing a carcinoma of the sigmoid within a year); one of diabetes; one of cystitis.

No case of recurrence of the caruncles has been described in the histories, which for the most part are filed in such a way that readmissions for any cause would be entered on the original histories. As noted above, in Case 2 the tissue studied was the second caruncle removed. This seemed to have occurred in only one other case studied.

PATHOLOGICAL FINDINGS

We have previously mentioned the fact that the epithelium which is found near the meatus may be of a fairly characteristic stratified or transitional type. In our 23 cases, 10 were found in which each type seemed predominant, while in 3, both were found in about equal proportions. This factor would be expected to vary depending on the direction in which the section was cut and the part sectioned.

With regard to the aforementioned apparent epithelial "infolding," there were 6 cases in which this was found in notable amount. Of these, 5 were tabulated as specimens in which transitional epithelium predominated. The apparent displacement of the cells downward was probably the feature which aroused the suspicion that the growth was not definitely benign. The absence of definite invasion of the subjacent structures, the uniformity as regards size and appearance of the epithelial cells, and the fact that for the most part they were properly oriented were all features which should dispose of these suspicions. The clinical data in our own cases and those reported in the literature and the extreme rarity of primary epithelioma of the female urethra further supported the position that these were benign conditions.

In 17 of the 23 cases, a definite glandular mass was present in the submucosa. These were acinar in form, and their inner lining was made up of a single layer of columnar cells. One is depicted in Figure 3. In our opinion these glands could not be confused with any other structure, and we believe them to represent glands of Skene. The high percentage of these glands seems to suggest that they may be an important factor in the formation of caruncles. This possibility seems the more likely when it is considered that the glands of Skene and the caruncles both occur, in almost all, if not all, cases in the posterior urethral quadrants.

Transitional epithelium predominated in 9 of the specimens in which the glands were found, stratified epithelium in 6, and both in equal amounts in the other 2.

Chronic inflammation was the rule, definite round cell infiltration of the stroma being found in 21 of the 23 cases. At least moderately enlarged vessels were found in 14 of the specimens.

SUMMARY

1. The epithelium of caruncles of the female urethra frequently shows enough infolding to make their actually benign nature appear doubtful to one who is not familiar with this particular structure.

2. Compound acinar glands similar to those described by Skene are frequently present in the female urethra. They are found in 17 of our 23 cases.

3. It is suggested that these glandular structures may be an important factor in the formation of caruncles.

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BRAIN-FLAP—ITS RELATION TO INTRACRANIAL PRESSURE

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THE work here reported is a continuation of that described in our earlier paper (5). At the time of its completion we had been unable to obtain results sufficiently consistent to determine what might be the relationship between intracranial pressure and the appearance of the extensive movements of the brain and dura mater with respiration which we have termed "brain-flap." This whole question, therefore, was left open pending further experiment. We had, however, observed in an early endeavor to lower intracranial pressure that the largest cerebral movements of which we had record occurred in tracings made after the intravenous administration of a hypertonic solution. This and other methods of altering intracranial pressure have given us clear and consistent results in the further investigation of brain-flap which will be described in the following pages.

CLINICAL EXPERIENCE

Before these further experiments had been performed, an opportunity occurred of observing in man the association of brain-flap with a reduction of intracranial pressure.

During a cerebellar exposure made by one of us (A. K. H.) for tumor, the occipital dura mater was found to be very tense and quite immobile. To relieve the tension before opening the membrane, a serum needle was passed into the hinder part of one lateral ventricle and was left there to drain off the cerebrospinal fluid. The flow from the needle was forcible at first, but afterward issued in drops which were more frequent when the patient strained. Within a few minutes from the time of the ventricular puncture and before the slackened dura mater had been opened, brain-flap made its appearance and soon increased to large dimensions.

The operation was performed under local anæsthesia with 0.5 per cent novocain, and throughout the procedure the patient talked or grumbled almost continuously. He had no pain but he complained of his position on the operating table—the usual face-down position with the head and shoulders supported—and though he suffered from no respiratory difficulty, respiration, none the less, was constantly modified throughout by his grumbling commentary.

The act of opening the dura mater did not increase the range of the movements of brain-flap but it clearly revealed the large excursions of the cerebellum, which bulged into the wound on expiration and receded deeply with each inspiratory act. These movements were seen for about half an hour and continued at a maximum until they were hidden by the suture of the muscular planes at the close of the intervention.

The level of the systolic blood pressure, recorded at intervals of 5 minutes from the brachial artery, remained remarkably constant. It is noteworthy that it stood at 110 millimeters of mercury (a) before the appearance of brain-flap, (b) at the time of its appearance, and (c) during almost the entire period in which maximal excursions of the cerebellum were observed. At the close only of the operation, the blood pressure fell to the level of 95 millimeters of mercury.[1]

EXPERIMENTAL METHODS AND RESULTS

Broadly speaking, the methods employed for recording the blood pressure and variations in the intracranial volume, etc., have remained unchanged from those described in our former paper. A few slight differences of detail exist. The volume recorder used for the present work has a sensitivity almost exactly half that of the instrument used before. A movement of one centimeter of arc corre-

[1] This case was briefly mentioned in a footnote to our previous paper (5).

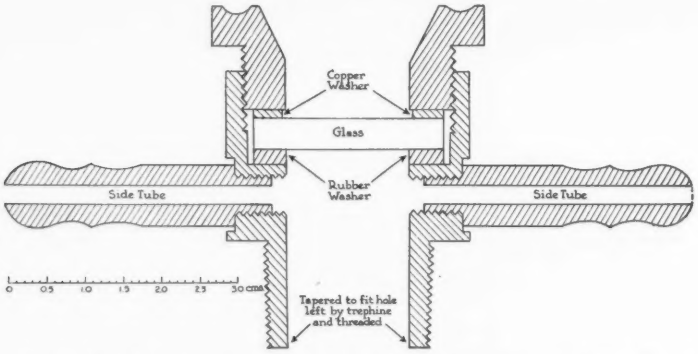

Fig. 1. This is a scale drawing of one of the brass instruments used. Taken in conjunction with the description in the text, it is self explanatory and no further details need be given.

sponds to a change of volume of 0.1 cubic centimeter.

The single brass instrument, described before, which was screwed into the trephine opening, has been replaced by a pair of improved design, made specially for this work by Mr. S. W. Bush of the Sir William Dunn Laboratory of Pathology, Oxford.

A scale drawing of one of them is shown as Figure 1. Each consists of four pieces of brass: One of these—the largest—was bored throughout its length and tapered at one end so as exactly to fit into the conical hole left by the trephine. On this taper a screw thread was turned. At its other end, the brass piece was bored out to a considerably larger diameter, and on the inner surface of this wider portion a screw thread was turned. At opposite points on the cylindrical outer surface, two holes were drilled and tapped, and into these were fitted two side tubes. On the shelf left in the first piece where the bore had been widened, were fitted, first, a rubber washer, then a circular piece of glass, and finally a copper washer. The fourth piece of brass was turned to fit the screw thread in the wider part of the first piece. When this fourth piece was firmly screwed home into the first there resulted a completely air-tight joint, thus leaving a potentially closed cavity below the glass, communicating with the external air by the two side tubes. The glass plate enabled one to observe the movements of the dura mater.

The experiments to be described fall naturally into three groups, under five headings: the first group dealing with lowering of the intracranial pressure by two different methods, the second with raising that pressure—again by two different methods,[1] and the third with the results produced by the sudden reduction of an artificially created high intracranial pressure.

A fourth group, totally unconnected with the others, has been added to complete the picture given in our earlier paper of the relations between brain-flap and arterial pressure.

The Effects of Lowering the Normal Intracranial Pressure

The results of hæmorrhage. It was shown by Dixon and Halliburton and confirmed by Becht that the withdrawal of a considerable volume of blood from an animal was followed by an immediate reduction of the intracranial pressure.

This simple procedure was clearly the easiest method of testing the effect of a reduced intracranial pressure in the production of brain-flap, there being nothing more than a mechanical change in that pressure to be considered (1). Using this method, we found by experiment that tracheal occlusion could elicit brain-flap in animals previously refractory, almost immediately after removal of from 10 to 15 per cent of their blood. This may be well seen in Figure 2.

The results of injection of 30 per cent solution of sodium chloride. It was first shown by Weed and McKibben, and their work has been confirmed by many others, that the intravenous injection of

[1]It was remarked by Hill that, in theory at any rate, a high intracranial pressure would tend to damp out, by merely physical means, any changes in brain volume, whether caused by variations in the conditions of the respiratory or circulatory system.

strongly hypertonic solutions produces, after a transient rise, a prolonged considerable decrease of intracranial pressure.

Our earlier experiments, using this method of reducing intracranial pressure, gave only the most inconsistent results and this was due, in our opinion, to insufficient dosage.

In the later part of our work, however, we have succeeded in procuring brain-flap on every occasion when a sufficient quantity of 30 per cent sodium chloride solution has been injected. In Figure 3 is shown a tracing which displays the effects of such an administration on the blood pressure, and at the same time, the *primary* increase in brain volume which corresponds exactly to the primary increase of intracranial pressure described by Weed and McKibben. The total amount injected was in the proportion of 4 cubic centimeters of the 30 per cent solution for each kilogram of body weight given in amounts of 4 or 5 cubic centimeters at intervals of about 1 minute.

The tracing shown in Figure 4 is taken from another animal, and it includes the entire period of an injection of 50 cubic centimeters of 30 per cent sodium chloride solution. It will be seen that, at the commencement of the experiment, no brain-flap resulted from closing the tracheal cannula. The injection of the solution was then commenced and the changes in blood pressure and brain volume seen in Figure 3 are again exhibited.[1] (Unfortunately blood pressure changes are not so well shown as in the previous figure owing to the clotting of blood in the arterial cannula.) Within a very short space of time after the injection had been completed, occlusion of the tracheal cannula produced the condition of brain-flap. The actual movements of the lever were not then extensive but became progressively greater as the experiment continued. This is seen in Figure 5, which shows a tracing taken from the same dog 35 minutes after the injection was completed. In the latter part of this tracing, movements of the brain-volume lever will be observed, which are marked "spontaneous." The hyperpnœic respirations, following on the preceding period of occlusion of the tracheal cannula, were now sufficient to produce a sufficient variation of thoracic pressure to elicit brain-flap. Occasionally there were seen, superposed on these "spontaneous" movements, very small variations in the brain volume which were due to individual heart beats. These were never observed in the normal tracing.

The Effects of Raising the Intracranial Pressure

The results of the intravenous injection of distilled water. Weed and McKibben were the first to show

[1]It must be stated most emphatically that the sustained rise of the brain volume lever seen in Figure 3 is deceptive; it is not due to a persistent increase of brain volume. The height of the lever is maintained by a slight hæmorrhage into the brass instrument. In our experiments this hæmorrhage almost invariably accompanied the transient increase of intracranial pressure which is produced as a primary effect by the injection of hypertonic solutions. The secondary and main effect—the fall of intracranial pressure—is, of course, accompanied by a decrease of brain volume, and this the lever does not record in Figure 3.

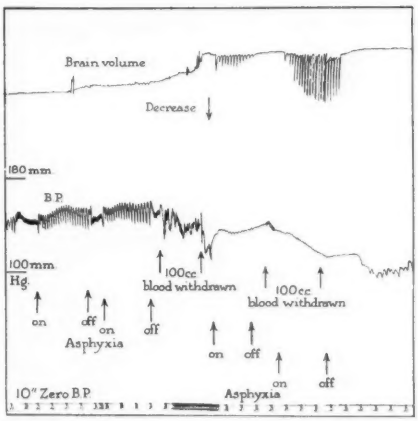

Fig. 2. This tracing displays the results of hæmorrhage. The withdrawal of 100 cubic centimeters of blood immediately produces a condition in which "asphyxia" is associated with brain-flap in an animal which, about 5 minutes earlier, had been entirely resistant. The further removal of a second 100 cubic centimeters of blood renders the movements of brain-flap progressively larger as the hæmorrhage proceeds.

that the intravenous injection of hypotonic solutions is followed by an increase of intracranial pressure, and their observations have frequently been confirmed by others. Again, as with the injection of strong solutions of sodium chloride, our earlier experiments to determine the relationship existing between brain-flap and injections of distilled water gave only inconsistent results and this, too, by reason of inadequate dosage.

More recently, however, we have obtained perfectly consistent results after the intravenous injection of distilled water in a dosage of from 40 to 45 cubic centimeters per kilogram of body weight. The procedure adopted was to prepare the animal in the usual way and then to remove from 10 to 15 per cent of its blood volume. As is described above, this renders the animal sensitive to occlusion of the tracheal cannula so that brain-flap occurs. When this condition had been obtained, distilled water, to the calculated amount, was run through a burette into the femoral vein just so rapidly as to cause only the slightest disturbances in the level of blood pressure. Within a very short time from the completion of this injection, the act of occluding the tracheal cannula produced only minimal signs of brain-flap. A tracing to demonstrate these points is shown in Figure 6.

In some experiments, the whole amount of water was not injected at once, but about two-thirds was first run into the vein and the animal was then tested for brain-flap. A tracing is reproduced as Figure 7 to demonstrate this. It will be seen that, after the first injection, there is a short period in which the

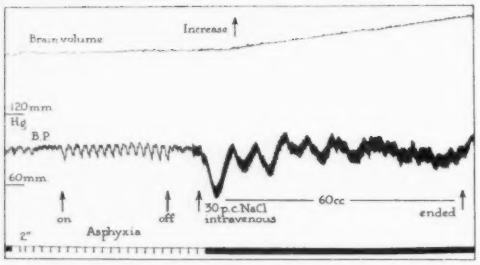

Fig. 3. In this tracing may be seen the *primary* result on brain volume of the intravenous injection of 60 cubic centimeters of 30 per cent sodium chloride. The effects on blood pressure are also seen. During the administration there is a progressive increase of the brain volume. The solution was run into the femoral vein in amounts of about 4 cubic centimeters at a time at intervals of about one minute. In the earlier part of the administration, each injection is followed by a marked drop in blood pressure but later this pressure is maintained.

movements of brain-flap are very small but that soon they return again to a comparatively large size. On the further injection of water, however, they are almost completely abolished and so remain for the rest of the experiment.

The effect of the mechanical application of an increased pressure to the brain. For these experiments the preparation of the animal was varied in that it was trephined on both sides of the skull. Into each hole was screwed one of the two brass instruments (Fig. 1). Of these, one was connected as usual to the volume recorder, the other—by one of its side-tubes—to a source of air pressure and to a water manometer. The air pressure was obtained by means of two aspirator bottles, each about half filled with water. These were so connected that, by raising one of them, a pressure could be exerted on the brain, the degree of which was measured by the water manometer. The air pressure on the brain could be reduced instantly to atmospheric level by opening the spring clip which closed the rubber tubing placed on the second side-tube of the same instrument to which the source of pressure and the manometer were attached.

Brain-flap was induced after withdrawal of blood, and when the condition was well established the effects of external pressure were examined. At first and for some considerable time, only the most inconsistent results were obtained. In two experiments, in which, so far as we could observe, conditions were identical, we would obtain results diametrically opposed. In one it would be found that the movements of brain-flap were completely suppressed by a pressure of 9 centimeters of water, while in the other the application of a pressure of 20 centimeters of water would only partially arrest them. It was by no means clear what the source of these inconsistencies could be. Finally, it was thought possible that there might be some variation in the firmness of the attachments of the dura mater to the skull. It may be stated in parentheses that, in the dog, these attachments over the parietal region may be fairly strong. As a result of these considerations, in the later experiments the effect on brain-flap of a pressure applied externally was examined (a) with the dura mater untouched, (b) with the dura mater freed from its attachments to the skull, and (c) with the dura mater opened. Figures 8, 9, and 10 have been prepared to illustrate these experiments.

In Figure 8 is shown the effect of the application of a pressure of 5 centimeters of water to the untouched dura mater. The brain volume lever instantly rises slightly[1] and the excursions of brain-flap are merely reduced. In Figure 9, the results of the application of a pressure of 10 centimeters of water are shown. The excursions of brain-flap are here virtually abolished.

In Figure 10 is shown a composite tracing in three parts, a, b, and c. Here a pressure of 9 centimeters of water was applied in each case. In Figure 10a there is only a small rise of the brain volume lever and little reduction of brain-flap, the dura mater having been left untouched. In Figure 10b, the rise of the brain volume lever is more marked and the movements of brain-flap cease almost completely. In this case the dura mater was freed from the skull. The degree of difference in the appearances in Figure 10c, with the dura mater opened, and those in Figure 10b with the membrane freed but intact is not great.

The Effect of the Sudden Release of a Considerable Degree of Pressure Applied Externally to the Brain.

For these experiments the preparation of the animal was exactly similar to that described in the last section except that no blood was withdrawn. The animal was first tested for brain-flap which was found to be absent. Pressure was then applied to the dura mater to a degree which would not affect the medullary centers. At intervals of about 10 minutes the tracheal cannula was occluded, without eliciting any appearance of brain-flap. When the increased pressure had been maintained for an hour, the air entry was again blocked, and again no brain-flap was produced. While powerful respiratory movements were still continuing, the pressure was suddenly lowered to atmospheric level by opening the side-tube of the brass instrument. There was then an instantaneous development of brain-flap. At intervals of 5 minutes for a further period of half an hour the animal was again tested for brain-flap, and it was found that the excursions of the lever became gradually smaller. Figure 11 has been prepared to demonstrate the instantaneous appearance of brain-flap when the external pressure is suddenly reduced.

The Effects of Brain-flap on the Pressure in the Circle of Willis

This part of the experimental work has no connection with the foregoing but was performed in

[1] This rise of the brain volume lever, when external pressure is applied, is not due to a real increase of brain volume. It is produced by the bulging of the brain at the trephine hole opposite to that where the brain is compressed.

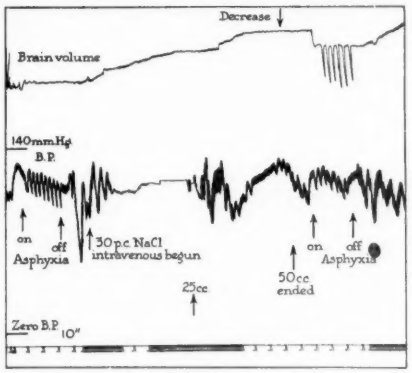

Fig. 4. This tracing is continuous, showing the whole period of injection of 50 cubic centimeters of 30 per cent sodium chloride. In the first part, "asphyxia" is not associated with brain-flap. There is the same increase in brain volume and effects on blood pressure as in the previous tracing. Immediately after the injection has been completed "asphyxia" produces brain-flap. The sustained height of the brain volume lever does not imply a sustained increase in brain volume. Its maintenance is due to hæmorrhage into the brass instrument which accompanied the primary increase of brain volume (see also the legend to Figure 11).

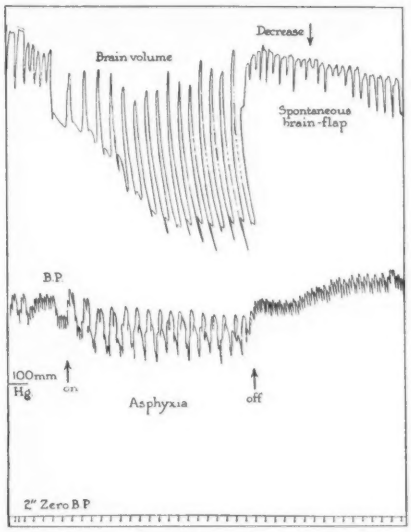

Fig. 5. This tracing was prepared from the same animal as that used for Figure 4. It shows exceedingly large movements of brain-flap during a period of "asphyxia." After the release of the tracheal cannula, the animal had very marked hyperpnœic respirations, which caused aspiration from the brain of sufficient blood to give rise to smaller movements of brain-flap.

order to complete the picture, given in our earlier paper, of the relations between brain-flap and arterial pressure. A cannula was inserted into the cephalic end of one of the carotid arteries and connected to a mercury manometer. In this manner a tracing was prepared illustrating the changes of blood pressure in the circle of Willis which occur during the production of brain-flap. This tracing showed, as had been expected, that these changes closely reproduced the ordinary asphyxial variations recorded in the femoral artery, though the actual size of the variations was smaller. In Figure 12 is shown a tracing displaying these observations.

DISCUSSION

It will, perhaps, be advisable first to consider a simple analogy. Let us suppose that an almost inextensible watertight bag has been made, provided with (1) a means of connection to a source of water under pressure, (2) a valve which can be closed but which, when opened, will permit a very small stream of water to escape, and (3) a cylinder with a very close fitting piston, which can be moved in and out through a short stroke (see Fig. 13). When the bag is filled with water under pressure, movements of the piston in and out in the cylinder—supposed to be small in comparison with the volume of the bag—will produce only very slight variations in the total amount of water in the bag itself and consequently there will be no appreciable movements of the tense wall of the bag. Now suppose that the needle valve be opened. A very small stream of water will escape and after a time the pressure within the bag will fall nearly to the atmospheric pressure. At this point, movements of the piston outward will bring down the pressure within the bag considerably and when this pressure falls, for a moment, below that of the atmosphere the wall of the bag will collapse inward.[1] The maximal excursion of the wall for any given length of stroke of the piston will occur when the pressure within the bag is exactly equal to the mean atmospheric pressure without.

Let us now apply this analogy of the bag to the skull contents. Here, too, there is an almost inextensible membrane, the dura mater, which represents the wall of the bag. For the sake of simplicity we shall consider only

[1] Naturally, under the conditions, there cannot be a permanent pressure within the bag which is less than that of the surrounding atmosphere.

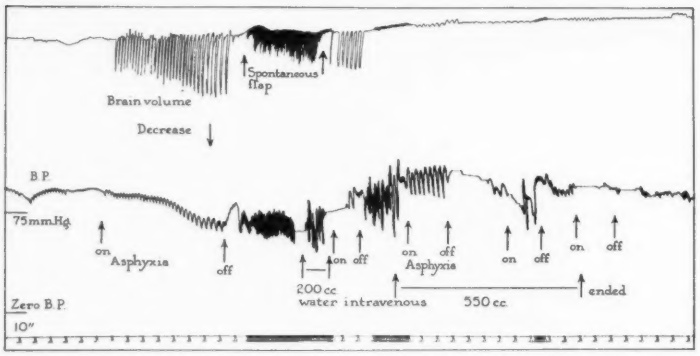

Fig. 6. This is a continuous tracing to show the effect of intravenous injection of water on brain-flap. The animal had been bled and the first part of the tracing shows brain-flap induced by "asphyxia," and even "spontaneous" movements due to pure hyperpnœa. Two hundred cubic centimeters were first injected, with the result that the brain-flap movements, consequent on "asphyxia," were reduced in size but not abolished. Very soon after the commencement of a further injection of 550 cubic centimeters of water, they were found to be practically nonexistent.

the movements of the dura mater. During life and in the normal subject, this membrane tends to bulge outward under intracranial pressure when an opening is made in the skull, and we may for the present purpose compare

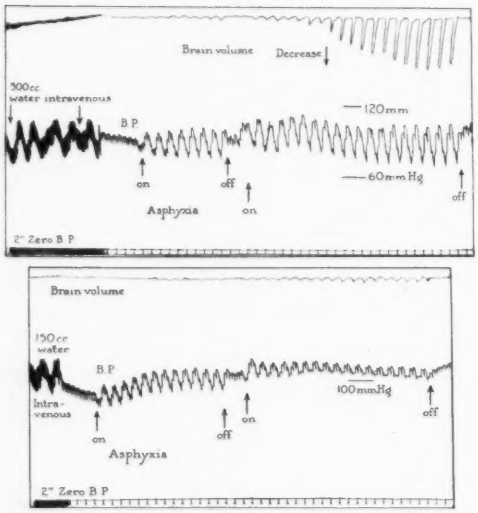

Fig. 7. This tracing is similar to the last. It shows a temporary abolition and subsequent reappearance of brain-flap after the injection of 300 cubic centimeters of water. The further injection of 150 cubic centimeters reduced the movements to a minimum and for the remainder of the experiment they remained so reduced.

the contents of the dura mater to the fluid contained under tension in the bag.

The actual movements of brain-flap were shown in our earlier paper to depend on the alternate acceleration and delay of venous outflow from the brain by a sufficient fall and rise of intrathoracic pressure—a fall and rise which is due to powerful respiration. The volume of the brain is in this way alternately diminished and restored. The thoracic pump thus corresponds to the piston and cylinder of the analogy, and it is clear that brain-flap—which is absent while intracranial pressure is at its normal, hyperatmospheric level—may be expected to appear when the mean intracranial pressure approximates to atmospheric pressure. Then, even a small aspiration of venous blood from the skull will, for a moment, bring intracranial pressure below that of the atmosphere, and the dural membrane will be pressed inward.[1]

Alternatively, a small rise of intracranial pressure, due to a delay of venous outflow from the skull, will now suffice to bulge out the almost equipoised membrane. It is thus obvious that the swing of the dura mater must—for a given cycle of the thoracic pump—reach its maximal amplitude when the mean

[1] Here, too, as was shown by Weed and Hughson, there can be no *permanent* reduction of pressure within the membrane below the level of atmospheric pressure.

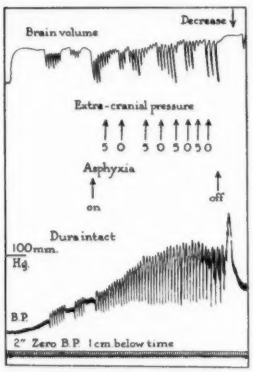

Fig. 8. This tracing demonstrates the effect on brain-flap of exerting an external pressure on the dura. The application of a pressure of 5 centimeters of water to the dura immediately reduced the amplitude of the excursions by about one-half, with a prompt return to their previous size on releasing the pressure.

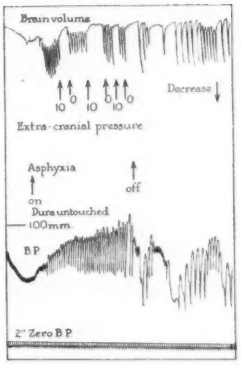

Fig. 9. This tracing was prepared from the same animal as Figure 8 with the dura still intact. It shows that with double the pressure—10 centimeters of water—the movements of brain-flap are virtually abolished.

intracranial pressure and the pressure of the atmosphere are precisely equal.

The experimental results described above bear out in unmistakable fashion the conclusions reached on theoretical grounds. In one group of experiments a reduction of intracranial pressure is followed by the appearance of brain-flap, and in another, the condition is abolished by increasing that pressure. In the third group, sudden reduction of a continuous pressure applied externally is associated with the immediate appearance of brain-flap.

The first group of experiments comprises those concerned with a reduction of the intracranial pressure, and the first method we adopted to effect this was the removal of blood from the animal. It was shown by Dixon and Halliburton, and confirmed by Becht, that hæmorrhage produces a prompt reduction of the intracranial pressure. The latter worker also showed that, on the return of the withdrawn blood to the circulation, the intracranial pressure immediately recovered its former level. On the grounds of these experiments, he attributed the reduction and subsequent restoration of that pressure to purely mechanical causes, and there seems to be no reason whatever for disputing his conclusions.

Reference to Figure 2 will show our experimental findings. In this case, occlusion of the tracheal cannula in the earlier part of the experiment produced no signs of brain-flap, in spite of vigorous respiratory efforts. Within a few moments after the withdrawal of blood, interference with the air entry to the lungs produced the condition. A further removal of blood, performed while the tracheal cannula was closed, was again associated with movements of brain-flap, which became progressively larger as the blood was withdrawn. Since it is accepted that hæmorrhage lowers the intracranial pressure, by far the easiest explanation of these experimental findings is that the appearance of brain-flap is here conditioned by two factors: (1) the alternate aspiration and delay of blood from the brain, produced by strong respiratory movements against an artificial resistance to the air entry, and (2) the presence of a lowered intracranial pressure.

The results obtained after the intravenous injection of strongly hypertonic solutions entirely support the conclusions arrived at above. In Figure 4 is shown a continuous tracing, typical of those obtained by the injection of 30 per cent sodium chloride. Before the injection was started, the animal showed no signs of brain-flap on occlusion of the tracheal cannula. Within a few seconds of the termination of the injection, resistance to air entry was associated with brain-flap. As time went on, further occlusion of the tracheal

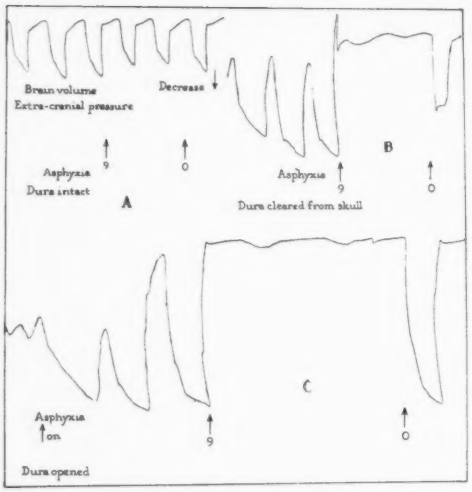

Fig. 10. This is a composite tracing. (The blood pressure curve has been omitted to economize space.) There are three portions, all prepared from one animal. A water pressure of 9 centimeters was applied. In A with the dura intact, only a small diminution of the movements occurs. In B with the dura intact but freed from the skull, there is an apparent increase in brain volume and an almost complete arrest of the excursions. In C with the dura freed and opened, there is a greater apparent increase in the brain volume with an equally complete abolition of the excursions. In all, there is an immediate restoration of the size of the excursions on releasing the pressure. (The apparent increase in brain volume is produced by the bulging of the brain at the trephine hole opposite to that where the brain is compressed.)

cannula produced progressively larger movements of the brain volume lever. This may be seen in Figure 5, a tracing taken from the same animal that was used for Figure 4. In the latter part of the tracing will be seen small movements of the lever, occurring when there was no resistance to breathing. These small movements are due to relatively small fluctuations of the intrathoracic pressure, caused by vigorous hyperpnœic respirations which follow the period of asphyxia.

To Weed and McKibben is due the credit for the observation that intravenous injection of strongly hypertonic solutions is followed by a reduction of the intracranial pressure. Their work has since been confirmed and expanded by many others (2, 4, 7). They attributed, and doubtless correctly, the fall of pressure to a depletion of the cerebrospinal fluid by the greatly increased osmotic pressure of the blood. They found, further, that intracranial pressure, after a brief rise, went on falling, until, in the animal with an intact skull, this pressure might be well below that of the atmosphere. In a later paper Weed and Hughson point out that such a reduction of the pressure below atmospheric level cannot occur in the animal with the skull opened.

Our experiments show that brain-flap makes its appearance very shortly after the commencement of the injection, at a time when, as found by Weed and McKibben, the intracranial pressure has begun to fall. Further, we find that the movements become progressively greater with the lapse of time. This close parallelism between the time relations, on the one hand, of the appearance and the amplitude of brain-flap, and, on the other, of the changes in the intracranial pressure, can be clearly explained by the dependence of brain-flap on these changes. It is interesting to observe that when intracranial pressure has been greatly lowered even the slight degree of acceleration and delay of venous outflow from the brain that occurs during pure hyperpnœa—with no resistance to breathing—is sufficient to cause small but unmistakable variations in the brain volume. This bears out the theoretical view of brain-flap, which commences this section, and necessitates a modification of our former conclusions, which we shall stress at the close of the discussion.

We come now to the consideration of the second group of experiments, in which, by two different methods, there was produced an increase of intracranial pressure. The first of these methods we again owe to the work of Weed and McKibben, who showed that the intravenous injection of strongly hypotonic solutions, such as distilled water, produced a rise in this pressure. The increase of intracranial pressure is, they believe, due to a change in the osmotic pressure of the blood, opposite, of course, in sign to that produced by hypertonic solutions. Their work has been confirmed frequently and their explanation remains unchallenged.

Figure 6 depicts most clearly the result of the intravenous injection of distilled water into an animal which had been so affected by

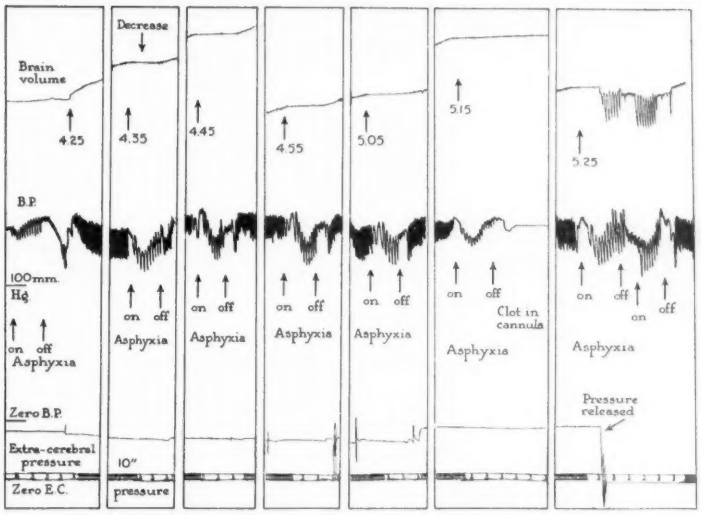

Fig. 11. This is a composite tracing, made up of small portions of a continuous tracing prepared in an experiment, in which a high pressure was maintained on the dura for an hour. At first, before the pressure was applied, no brain-flap resulted from "asphyxia." At 4:25 p.m. the pressure was applied and maintained for an hour, and during this period "asphyxia" was induced at 10 minute intervals without eliciting any trace of brain-flap. The pressure varied from 30 to 40 millimeters of mercury during the experiment. There is a steady rise in the level of the brain volume lever, artificially interrupted at intervals. This rise was due to a slow leak of blood from the skull into the cavity of the brass instrument. At 5:25 p.m. "asphyxia" was once more induced without eliciting brain-flap. During the maintenance of the "asphyxia," the pressure was suddenly released and brain-flap instantaneously appeared. Later, the movements became less, and finally disappeared after about half an hour. This is not shown in the tracing.

hæmorrhage as to show well marked brain-flap. The water was injected discontinuously in amounts of 50 cubic centimeters at a time. The immediate result of the administration of 200 cubic centimeters was a reduction of the amplitude of brain-flap. A further 500 cubic centimeters was then injected, the animal being tested for brain-flap at intervals. Very shortly after the commencement of this second injection, the condition failed to reappear and was not seen again during the remainder of the experiment. The cessation of brain-flap so closely follows the injection of water that it is difficult to escape the conclusion that disappearance of the movement is directly due to the rise of intracranial pressure induced by that injection. The tracing represented in Figure 7 completely supports this view.

The second method employed in the investigation of this part of the problem was the application of a definite but small pressure to the intact dura mater, the contents of which may, for our purposes, be considered as a fluid which will transmit pressure equally in all directions. The experimental difficulties met with have been explained and need not be further discussed.

In Figures 8 and 9 are shown two tracings prepared from the same animal, in which brain-flap had been induced by hæmorrhage. In the former will be seen the results of applying a pressure of 5 centimeters of water to the dura mater and in the latter the effects of a similar application of 10 centimeters of water pressure. With 5 centimeters there is an increase in the apparent volume of the brain and a marked diminution in the excursions of the brain volume lever. With 10 centimeters there is a similar apparent increase in the brain volume, but the movements of the lever

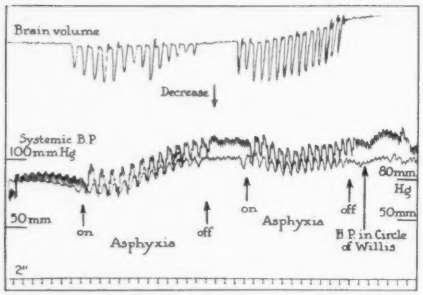

Fig. 12. This tracing displays the effect of eliciting brain-flap on the blood pressure in the circle of Willis. It is easily seen that the variations reproduce in miniature those occurring in the femoral artery. They are dependent therefore on "asphyxia" rather than on brain-flap.

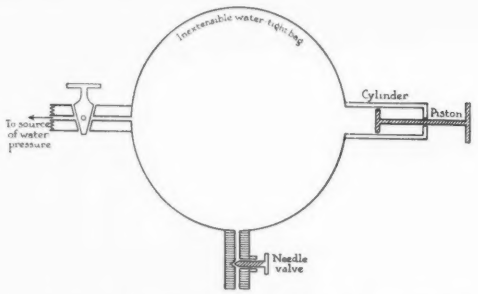

Fig. 13. This is a diagram prepared to simplify the appreciation of the theoretical part of the discussion. It requires no further description than appears in the text.

cease almost completely. In each case, reduction of the external pressure to atmospheric level is followed by complete restoration of the amplitude of the excursions. These experiments, we think, are absolutely conclusive. There can be no doubt that the increase of intracranial pressure produced by the application of external pressure to the dura led, in the one case, to a reduction and, in the other—with double the pressure—to a virtual abolition of brain-flap.

Our third group of experiments, represented in Figure 11, must now be considered. It was thought that the application of a considerable degree of pressure to the dura mater would in time drive a certain amount of cerebrospinal fluid—together with venous blood—out of the skull by way of the veins. Fluid would also pass directly to the spinal subarachnoid space through the foramen magnum. This depletion would reduce the volume of the cranial contents, and when the external pressure was removed intracranial pressure would fall. To test the relation of this fall to the appearance of brain-flap we chose an external pressure of from 30 to 40 millimeters of mercury, which could be applied to the dura mater without causing any appreciable change in blood pressure. The tracheal cannula was occluded before the experiment, and at intervals of 10 minutes throughout its duration, without eliciting cerebral movements. Finally, after the application of pressure had continued for an hour, the cannula was again closed, and while the animal was making vigorous inspiratory efforts the pressure was suddenly removed. There was then, as we anticipated, an absolutely instantaneous appearance of brain-flap.

It remains now to consider how far the clinical evidence supports the experimental. In the case described in the present paper, the cerebrospinal fluid was drained off by a needle from the ventricles. The flow was rapid at first but later became slow. This slowing can be attributed only to a reduction of the intracranial pressure toward atmospheric level. Brain-flap here was exceedingly well marked. There was no external interference with the respiration but the continuous conversation of the patient must have placed some obstruction in the path of a free air entry.

In the three cases reported in our former paper, in which a prolonged period of brain-flap was present, there was in each a draining off of the cerebrospinal fluid. In Wheeler's case, there was a fracture of the base of the skull and the cerebrospinal fluid was seen escaping through the nose. In McConnell's case, a hydrocephalus was tapped. In our own case, the lateral ventricle had been drained.

These four clinical cases—the only ones in which a prolonged period of brain-flap has been observed—resemble our experimental cases in which the intracranial pressure had been greatly reduced.

In the discussion of our experimental cases we have sought to prove, and in our opinion have proved, that, for the appearance of brain-flap, a reduced intracranial pressure is necessary. When these clinical cases are taken into

consideration, it is obvious that they clinch our deductions.

The other clinical cases, in which a short period only of brain-flap was observed, may be correlated with the experiments in which a high external pressure was applied to the dura mater for some time and then suddenly released. In each of the clinical cases, a piece of bone which depressed the dura mater was removed, and, as soon as the membrane was relieved of this external pressure, a few large movements of brain-flap were observed, which died away rapidly. In our experimental work a high pressure—30 to 40 millimeters of mercury—was applied to the dura mater and left to act for an hour. Synchronously with the sudden removal of this pressure during a period of dyspnœa, brain-flap at once began. The condition was not permanent, and, after about half an hour, could no longer be demonstrated. We have no means of estimating what degree of pressure was being exerted by the depressed bone in the cases of fracture, but, judging by the similarity of effect, it would seem that the causation of the brain-flap, both in the clinical cases and under experimental conditions, was precisely similar, since in each category the first appearance of cerebral movement exactly synchronized with the relief of pressure, and, in each, the movement was transient.

Finally, it will be necessary to modify one deduction made in our previous paper. We have observed in our further experiments that brain-flap may occur during a period of hyperpnœa when there is no abnormal resistance to respiration. We think, therefore, that we laid too much stress on the need for this abnormal resistance as a factor in the causation of brain-flap. On the other hand we are most firmly convinced that brain-flap is conditioned by variations of intrathoracic pressure. It is clear, too, from the analogy of the bag, that—given a sufficiently low intracranial pressure—even a small aspiration by the thorax of venous blood from the brain will produce a definite, though small, diminution in brain volume.

SUMMARY

1. A further study is presented of the large respiratory excursions of the exposed brain (and dura) which the authors have termed "brain-flap."

2. A case of prolonged brain-flap in man, associated with great reduction of intracranial pressure, is described.

3. A series of experiments designed to show the relationship between brain-flap and the intracranial pressure, is described and discussed.

4. It is proved (a) that brain-flap is inhibited by factors which raise intracranial pressure; (b) that it is directly associated with factors which lower intracranial pressure.

5. It would appear from clinical and experimental observations that brain-flap does *not* occur when the intracranial pressure is normal.

6. The appearance and disappearance of brain-flap is explained on simple physical grounds, thus:—

7. The wall of a membranous bag which is already tense with fluid contained under a pressure greater than that of the atmosphere will not show obvious oscillation if *small* additions to the fluid contents alternate with *small* subtractions. Oscillations of the membranous wall due to these small additions and subtractions become obvious only when the mean pressure within the bag approaches the external pressure.

The membrane will be most sensitive to change of pressure, and its movements will be maximal, when the mean pressure on its inner face is equal to that which the atmosphere exerts on its outer surface.

8. Brain-flap thus appears if, and only if, the mean intracranial pressure approximates to that of the atmosphere. Then: (a) the exposed dura mater (or brain) is pushed inward by the atmosphere whenever the fall of intrathoracic pressure, which accompanies inspiration, withdraws enough venous blood from the skull to bring the pressure of its contents, for a moment, below the level of atmospheric pressure. (b) The sunken dura mater is pushed outward when expiration—by raising the intrathoracic pressure—checks the venous outflow from the skull so that intracranial pressure begins to rise.

9. The excursions of the exposed brain (or dura mater) become maximal when the *mean*

intracranial pressure is equal to atmospheric pressure, in the same way that—during a clinical estimation of diastolic pressure—the arterial wall moves most with each heart-beat whenever the mean pressure within the artery is balanced by the pneumatic cuff.

10. Further experimental work has shown that hyperpnœa, in the absence of respiratory obstruction, is sufficient to induce some degree of brain-flap, provided always that the intracranial pressure is sufficiently low.

11. During a period of brain-flap, the changes in blood pressure in the circle of Willis do not differ from those which occur at the same time in other parts of the systemic circulation; they are an exact reproduction in miniature of the pressure changes in the femoral artery, and, like them, are asphyxial in origin.

The authors desire to place on record an expression of their gratitude to Dr. K. Samaan, M.Sc., Ph.D. (Manchester), for his most kind assistance in the performance of these experiments.

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CHANGES IN THE SPINAL FLUID FOLLOWING INJECTION FOR SPINAL ANÆSTHESIA

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SPINAL anæsthesia produces less toxæmia and almost negligible protoplasmic disturbance as compared with general anæsthesia because of the supposed non-toxic action and small amount of the medicament used. However, there are certain immediate and remote sequelæ to spinal anæsthesia which as yet have not been satisfactorily explained. Munchmeyer states that in 1,189 cases of spinal anæsthesia with stovaine, not more than 70 per cent were entirely free from by-effects or after-effects. One patient in his series died from respiratory failure and seven suffered transient paralysis. Schultz reports a series of 2,251 cases operated on by means of apothesine spinal anæsthesia. In no case was there a mortality attributed to the effects of the anæsthesia. However, vomiting or retching occurred in practically every case and other sequelæ, such as incontinence of urine and fæces, headache, and paræsthesias of the leg, were common, all of which disappeared spontaneously. Three patients complained of pain along the course of the sciatic nerve from 1 to 4 weeks after the operation. Madden reports 3 fatalities following the use of stovaine spinal anæsthesia in doses of from 40 to 60 milligrams. Failure of respiration was given as the cause of death. Heinke and Laewen have observed severe circulatory disturbances with weak pulse and pallor in 2 cases following novocain spinal anæsthesia in doses of 15 milligrams. Gabbett reports one fatality following a slight operation under spinal anæsthesia in which he used 100 milligrams of novocain and 1 milligram of strychnine. In this case death also resulted from respiratory failure. Hatcher and Eggleston, in discussing the toxic action of novocain, state that they have been informed of two deaths resulting from novocain spinal anæsthesia. In one case the patient rolled off the table in the state of opisthotonos and died of failure of respiration. Beraud reports a fatality in a male 60 years of age, apparently in excellent physical condition before operation after stovaine spinal anæsthesia. Death in this case was attributed to cardiovascular embarrassment.

Le Poutre cites a case of urinary incontinence with perineal anæsthesia in a young man of 23 after spinal anæsthesia and suggests that the irritating action of the medicament

employed and the degree of concentration may be important factors in the sequelæ following spinal anæsthesia.

Smith and Porter studied the effect of the spinal anæsthesia on the blood pressure in cats and found that in only 1 of their series of 20 was there a marked fall in blood pressure, which they attribute to paralysis of the bulbar vasomotor center. They also found that a greater fall of blood pressure occurred in cases in which adrenalin was used in connection with novocain. Gray and Parsons studied the blood pressure in man during spinal anæsthesia and found a slight fall which they claim was due to relaxation of the muscles of the abdomen and lower limbs; the greatest fall in blood pressure obtained in some cases was attributed to paralysis of the intercostal muscles and the consequent diminution in the pumping action of the chest.

Much has been written regarding the chemistry and cytology of the cerebrospinal fluid in morbid conditions where changes from the normal may be expected. It was thought that if some of the sequelæ of spinal anæsthesia were due to toxic and irritating actions of the medicament employed, changes in the spinal fluid would result. With this in mind the following investigation was undertaken.

A review of the available literature reveals the fact that some work of this kind has been done mainly by French investigators. Revault and Aubourg in 1901 showed an increase in cytology in the spinal fluid following the use of spinal anæsthesia with cocaine, which they interpreted as a meningeal reaction to the drug used. Quinard reported like findings, that is, a pleocytosis with the production of a fibrinous coagulate following cocaine spinal anæsthesia. Barker examined the spinal fluid 46 hours after spinal anæsthesia with stovaine and found a definite increase in the cytology, which consisted of "leucocytes of various forms." The extent of the pleocytosis and the number of cases examined is not stated. Sicard and Salin in 1910 showed a marked increase in cytology in the spinal fluid following the injection of horse serum, Ringer's solution, and sodium chloride respectively in animals. Mestrezat and Riche in 1911 studied 10 cases 6 to 12 hours after spinal anæsthesia with stovaine and reported a slight increase in sugar and chlorides, slight pleocytosis without an increase in the albumin content of the spinal fluid. They conclude that spinal anæsthesia with stovaine is harmless as far as the nerve centers are concerned. Krey studied the spinal fluids of 8 cases, 3 of which were after the injection of 5 per cent tropocain in saline. The others were after the injection of phenolsulphonephthalein and neosalvarsan. In the first few hours after injection of these drugs, there was a slight pleocytosis, mainly of lymphocytes, together with a slight increase in albumin. He makes no distinction between the findings with tropocaine and those with the other agents employed. More recent work by Mader shows that after the injection of Ringer's solution, there is a rapid increase in sugar and lymphocytes in the spinal fluid, with a gradual return to normal.

Leclerc made observations on the spinal fluid in 31 cases of spinal anæsthesia in which 5 per cent allocain (Lumiere) without adrenalin was employed. Punctures were made from the day following the anæsthesia up to the eighth day. In 9 of 31 cases there was observed a pleocytosis of about 15 to 21 cells per field, the cells all being lymphocytes. In one case there was a polynuclear increase in the cells. Increase in the albumin content of the spinal fluid occurred in 11 of 31 cases. However, the most constant finding in the spinal fluid was an increase in the sugar which occurred in 21 cases. In attempting to correlate the clinical symptoms and changes in the spinal fluid observed, Leclerc was not able to find any relationship between the increase in cytology and the occurrence of cephalalgia. In his series there were 2 such cases which did not show any increase in the number of cells over the normal. However, there were 6 cases with manifestations of mild meningitis (headache, vomiting, backache, sometimes rigidity of the neck). In these, pleocytosis occurred in 4 cases of 8, 15, 15.3, and 21 lymphocytes per field respectively. However, he was not able to correlate the other findings, such as increase in albumin and sugar, with the occurrence of post spinal anæsthesia sequelæ.

In this investigation, spinal fluids were obtained in the usual manner employed in the

TABLE I.—SPINAL FLUID CELL COUNT BEFORE SPINAL ANÆSTHESIA AND TWELVE HOURS AFTER OPERATION

Cell count per cubic millimeter spinal fluid

Case	Before	After
1	5 lymphocytes	60 lymphocytes
4	10 lymphocytes	25 lymphocytes
6	0	70 lymphocytes
8*	3 lymphocytes	12 lymphocytes
9	3 lymphocytes	5 lymphocytes
10	5 lymphocytes	800 polymorphonuclears
11	0	200 lymphocytes
14*	4 lymphocytes	12 lymphocytes
16*	3 lymphocytes	250 lymphocytes
17	0	20 lymphocytes
19*	5 lymphocytes	90 lymphocytes
20*	5 lymphocytes	240 lymphocytes
21	0	200 {50 per cent polymorphonuclears {50 per cent lymphocytes
30*	5 lymphocytes	120 lymphocytes
Average	3.43	150.2

*Cases manifesting postoperative spinal anæsthesia sequelæ (Table III).

TABLE II.—SPINAL FLUID SUGAR DETERMINATIONS BEFORE SPINAL ANÆSTHESIA AND TWELVE HOURS AFTER OPERATION

No.	Reducing substance Mgm. per 100 c.cm.		Difference, mgms.	Per cent rise
	Before	After		
1	68.0	88.0	20.1	29.4
2	88.0	115.0	27.0	30.6
3	100.0	125.0	25.0	25.0
4	71.0	88.0	17.0	23.9
5	83.0	103.0	20.0	24.0
6	78.0	84.0	6.0	7.6
7	80.6	115.0	34.4	42.6
8*	68.0	85.0	17.0	25.0
9	87.0	111.0	24.0	27.5
10	68.0	80.0	12.0	17.6
11	86.0	133.0	47.0	54.6
12*	62.5	78.0	15.5	24.8
13	69.0	89.0	20.0	29.9
15	68.0	160.0	92.0	135.2
16*	83.0	108.0	25.0	30.1
17	71.1	100.0	29.0	40.8
19*	78.0	85.0	7.0	8.9
20	62.0	105.0	43.0	69.3
21	71.0	76.0	5.0	7.0
23	65.0	125.0	60.0	92.2
Average	74.35	102.65	27.2	37.3

*Cases manifesting postoperative spinal anæsthesia sequelæ.

operating room just before the introduction of novocain into the canal. Part of the fluid was used as solvent for the anæsthetic and reinjected into the spinal canal. The remainder was sent to the laboratory and examined immediately. Twelve hours after operation, lumbar puncture was again performed, and an examination of the fluid made. The following tests were employed:

1. Cell count and differential white count.
2. Chemistry, which included the nitric acid ring test for albumin, the Noguchi test for globulin, and Fehling's test for reducing substances.
3. Colloidal gold reaction.
4. Quantitative determination of sugar by the Folin-Wu method.

These tests were done immediately after withdrawal on all spinal fluids. The patients were observed closely for the development of any neurological phenomena. A careful record of the occurrence of any untoward symptoms such as headache, etc., was also kept.

Thirty-one cases were studied in this manner. Of these, there were 14 in which the spinal fluid did not contain any red blood cells. Those fluids in which the presence of red blood cells was considered as due to trauma incidental to the lumbar puncture were not included in Table I. Of these 14 cases, 11 showed a definite increase in the white blood cell content, the highest being 800 per cubic millimeter, all of which on smear proved to be polymorphonuclear leucocytes. Four cases showed counts of 240, 250, 200, and 200 cells; on smear the first three being all lymphocytes, the fourth case showing 50 per cent polynuclear cells and 50 per cent lymphocytes. Six cases showed counts varying from 20 to 120 cells, all lymphocytes.

The remaining 3 counts were 5, 12, and 12 cells respectively, which here are not considered as a definite increase in the cytology. Thus, of the 14 cases, there were 11 showing a very definite pleocytosis, 2 a slight pleocytosis, and 1 a normal count (see Table II).

A study of the chemistry, i.e., the albumin and globulin content revealed no departure from the normal. The albumin was not increased and in no case was the globulin reaction positive. This would indicate that the protein content of the spinal fluid was not altered, at least qualitatively. This was confirmed by the colloidal gold reaction, since in no case was there any reduction of the colloidal gold.

In 20 of the 31 cases, the quantitative determination of sugar was done before the 12 hours after the injection of novocain. In all 20 cases there was an increase in the sugar content, in the postoperative specimens varying from 7 to 135.2 per cent. In but 3 cases (6, 19, 21) could the increase be considered

TABLE III.—POSTOPERATIVE SPINAL ANÆSTHESIA SEQUELÆ WITH SPINAL FLUID FINDINGS

Case	Symptoms	Spinal fluid			
		Cell count		Sugar—mgm.	
		Before	After	Before	After
8	Vomited three times 24 hours postoperative	3 w. b. c. Lymph	12 w. b. c.	68	85
12	Nausea first 24 hours postoperative	3 w. b. c. Lymph	800 r. b. c. 100 w. b. c. Lymph	62.5	78
14	Vomiting at end of operation	4 w. b. c. Lymph	12 w. b. c. Lymph		120
15	Vomiting first 24 hours postoperative	3 w. b. c. Lymph	70 r. b. c. 50 w. b. c. Lymph	68	160
16	Retching during operation. Vomited twice first 24 hours postoperative	3 w. b. c. Lymph	250 w. b. c. Lymph	83	108
19	Severe headache first 24 hours postoperative	5 w. b. c. Lymph	90 w. b. c. Lymph	78	85
20	Severe retching during operation. Vomiting during first 24 hours postoperative	5 w. b. c. Lymph	240 w. b. c. Lymph	62	105
23	Vomiting during first 24 hours postoperative	5 w. b. c. Lymph	140 r. b. c. 100 w. b. c.	65	125
24	Pain in right leg 2 days postoperative—duration 4 days. Inability to void first 24 hours postoperative	many r. b. c.	many r. b. c.		88
28	Vomiting, headache, dizziness, backache, pain in right leg	3 w. b. c. Lymph	2100 r. b. c. 20 w. b. c. Lymph		150
29	Nausea and vomiting during operation and 24 hours postoperative	3 w. b. c. Lymph	10 r. b. c. 30 w. b. c. Lymph		
30	Vomiting and headache first 24 hours postoperative	5 w. b. c. Lymph	120 w. b. c. Lymph		
31	Inability to void first 24 hours postoperative	3 w. b. c. Lymph	many r. b. c.		

within normal limits, being less than 10 per cent. In the 17 other cases there was a significant increase in sugar. The greatest increase occurred in case 15 where the content rose from 69 milligrams per 100 cubic centimeters of fluid before injection to 160 milligrams 18 hours after operation, a rise of 135.2 per cent. This estimation was repeated and confirmed. There was nothing unusual in this case to account for this rise, neither were there any unusual complaints or clinical findings present. The average rise calculated on the basis of percentage increase for each case was 37.3 per cent.

Of the 31 cases studied 13 developed mild postanæsthesia sequelæ such as nausea, vomiting, retching, headache, backache, inability to void, and sciatica-like pains in the leg (Table III). A study of the spinal fluid in these cases reveals the fact that in 6 there was a definite increase in the cytology of the spinal fluid viz.: 12, 12, 250, 90, 240, and 120 cells, all of the lymphocytic variety. The 7 other cases showed red blood cells in the spinal fluid which as stated above were attributed to the trauma incident to the tap.

However, those 7 cases which showed a definite pleocytosis (60, 25, 70, 800, 200, 20, 200 cells) after the spinal anæsthesia did not manifest any postspinal anæsthesia sequelæ whatsoever (see Table I).

The spinal fluid sugar in 10 of the 13 cases showing postanæsthesia sequelæ were definitely increased. In the 3 other cases determinations were not made. However, there were 16 cases which also showed very definite rises in the spinal fluid after the spinal anæsthesia, which did not present any sequelæ. There seems, therefore, to be no correlation between the occurrence of sequelæ after the spinal anæsthesia and the changes in the spinal fluid recorded above.

SUMMARY

1. The spinal fluids of 31 cases were examined 18 hours after spinal anæsthesia.

2. Of 14 cases 11 showed a definite pleocytosis, 10 cases being of the lymphocytic variety, 1 of the polymorphonuclear variety. Seventeen cases were not considered because of the presence of red blood cells in the spinal fluid.

3. In 20 of the 31 cases examined, there was an increase in the spinal fluid sugar averaging 37.3 per cent.

4. There was no change in the albumin, globulin, or colloidal gold curves.

5. Thirteen of the 31 cases developed mild postanæsthesia sequelæ.

6. There was no correlation between the occurrence of postanæsthesia sequelæ and the changes observed in the spinal fluid.

7. The technique of spinal anæsthesia caused in certain instances a mild meningeal reaction, but apparently does not produce serious organic changes, as reflected in the change of the composition of the cerebrospinal fluid.

CONCLUSIONS

Just what the cause of the increase in cytology and sugar in the spinal fluid after spinal anæsthesia is, we cannot say. The pleocytosis would seem to indicate a certain amount of irritation of the serous lining of the subarachnoid space, but whether it was due to a general irritation of the whole extent of the dura or only of the immediate neighborhood of the puncture, it is impossible to say from these observations on the human subject. The increase in sugar together with the increase in lymphocytes in the spinal fluid might be interpreted in the nature of a mild encephalitis (since spinal fluid findings in the latter condition are quite the same as those recorded above). However, the absence of sequelæ in more than half of the cases studied, as well as the rapid disappearance and the mild character of the symptoms present after spinal anæsthesia would argue against this.

Further studies are being made in which simultaneous blood and spinal fluid sugar determinations are being done, before and after novocain injection, in order to observe if any relationship exists between the sugar content of the blood and spinal fluid under these conditions.

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CLINICAL SURGERY

FROM THE DEPARTMENT OF SURGERY, WASHINGTON UNIVERSITY

MIRAULT OPERATION FOR SINGLE HARELIP

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EACH year in the United States approximately one thousand children are born with more or less complete patency of the lower part of the primitive frontomaxillary fissure. In the great majority of these children sufficient tissue is present, if properly used, to permit of an acceptable surgical repair. At least this is true of the defects of the lip and nose.

Because the lip defects are apparently simple clefts and because much of the dicta on their repair has inclined toward triteness, many surgeons have fallen into the gross error of thinking that the repair is easy, hardly worthy of special effort. Yet, in spite of these facts, in few other surgical situations are evident possibilities and average accomplishment so far apart.

The correction of such defects *is* more or less simple, but it is a simplicity that is attained only by grinding effort. Though these open clefts differ much in appearance in various cases, their individual differences are more of degree than of kind, so that any one of several operative plans can be made applicable to the ordinary case. If he learns to use any one of the standard methods, the surgeon who assays the correction of nose and lip defects will expend his energy to greater profit than if he attempts to exercise eclecticism, or more dangerous yet, to contrive new methods. It is true that operative skill rather than breadth of acquaintance will bring greater satisfaction to the patient thus afflicted. This statement is made with no intention of belittling invention or ingenuity but rather to urge that these qualifications be directed, in the case in hand, toward adapting some well tried out plan. As a rule the simple plans are easier to execute but they are less plastic. Every added complexity of technique is a distraction and is justified only by commensurate added possibilities. Before adopting the more complex methods, therefore, the operator should make himself familiar with every detail of the operation and should understand the logic for doing it. As the operator acquires more skill he may feel justified in adopting a method that in earlier days he considered less feasible, but each modification will be like changing a golf stroke—not always free from immediate grief.

After the surgeon has gained the greatest possible surgical and mechanical skill with the most congenial method, he may still find that the results are not really pleasing unless his technique includes also artistry, which here, as in portraiture, can camouflage a multitude of defects.

In the repair of the lip, an open alveolar cleft is one of the great "bugbears" but we believe this need not be so.

Plans for forcefully approximating the separated halves of the hard palate were discussed and dismissed as unnecessary in the first quarter of the nineteenth century, to be revived in the third, and to be popularized in the fourth quarter, until now the operation has come to be regarded by many as indispensable in the treatment of wide open clefts. A number of surgeons now secure a uniformly high average quality of repair of lips and palates following preliminary forceful closure of the alveolar cleft. Our own observation and experience, however, have convinced us that:

A. B. C.

Fig. 1. Replacement of the premaxilla. A, Diagram of an open, complete single cleft. B, Shows desired relationship of the premaxilla to the maxilla in the closure of the alveolar cleft. This gives the greatest prominence to the upper lip and least width to the anterior part of the palate cleft. Such a relationship usually follows within 3 to 15 months after a simple repair of the lip over the open alveolar cleft. C, Shows an improper adjustment of the premaxilla to the maxilla, resulting from misdirected force. It causes retraction of the upper lip and an increased width in the anterior part of the cleft palate. See also Figures 6, 7, and 8.

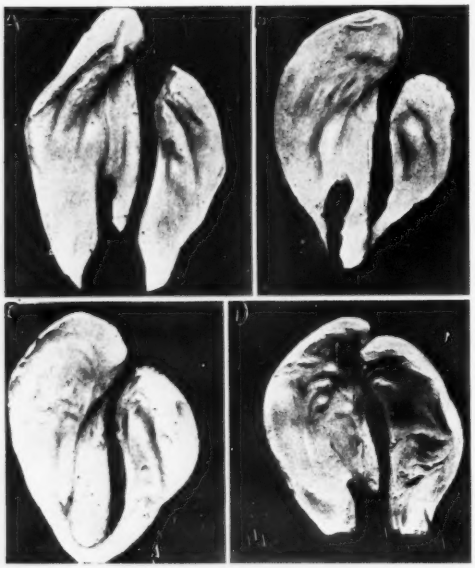


Fig. 2. Shows the closure of the alveolar part of the cleft that will usually occur when the lip is repaired. A, is a cast made of a cleft palate in a child 20 weeks old. At this age it is not good practice forcefully to close the alveolar part of the cleft, but the lip is repaired. The other three casts were made at 23, 30, and 44 weeks, respectively, and show the gradual closure which occurred from the lip pressure without any damage to the developing teeth. This closure by lip pressure will occur usually regardless of the width of the cleft.

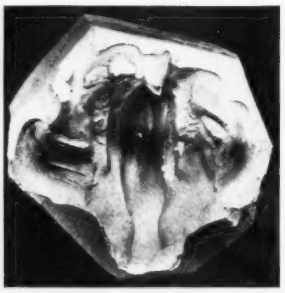


Fig. 3. Cast of a child whose lip has been repaired over an open alveolar cleft at 2 weeks of age, yet, 23 months later, it will be seen that there was a wide alveolar cleft. In the few cases we have observed in which the alveolar cleft failed to close in a reasonable time from lip pressure, approximation in this part has rapidly followed the flap operation on the palate but in young infants it is not practical to unite the flap edges along the anterior third of the bony cleft if there is a wide alveolar separation. This does not always necessitate an extra operative sitting because this part of the palate can be closed later at the same time as the opening in front of the alveolar process is closed.

A. B.

Fig. 4. Maxillary wiring, growth distortion. A, Dentures of an 11 year old child in whom a complete left sided cleft was closed by transfixing wires at the age of 2 days. Note the lack of forward progression of the maxilla and that the teeth of the lower jaw are inclined lingually. B, Shows the condition at 17 years of age after 6 years of almost continuous treatment by an experienced orthodontist. This is a better orthodontic restoration than has been obtainable in a number of such extreme deformities that have come under our observation. Though such deformities are the exception rather than the rule, their possible occurrence should, we believe, taboo the practice of transfixing the infantile maxilla with wires.

Fig. 5. Maxillary wiring, profile. Profile of Figure 4B showing flatness of the upper lip due to the lingual position of the upper incisor teeth, despite the orthodontic treatment. This flatness has forced upon us the practice of replacing these displaced anterior teeth with an artificial denture in order to give proper prominence to the upper lip.

1. Unless done with real skill and judgment, forceful closure of the alveolar cleft can add difficulty to rather than ease the subsequent mucoperiosteal closure of the palate cleft (Fig. 1).

2. Although the immediate results from the technique described by Brophy may be good, bad deformity of the upper jaw may subsequently develop (Figs. 4 and 5). In some instances such deformities can be corrected to some extent by years of orthodontic work but in others they can only be compensated for by most exacting surgical and prosthetic procedures (Figs. 6, 7, and 8).

Fig. 6A.

Fig. 6B.

Fig. 6C.

Fig. 6. Maxillary wiring, profile. A, Shows profile of a girl who, following early bone wiring, had an extreme retraction of the maxilla.

B, The dotted line shows the amount of correction that was obtained by a combination of orthodontia, the advancement of the cheeks, lip, and columella on their supporting structures, and the use of an artificial denture. All of this could have been avoided had the simpler plan of maxillary adjustment been used.

C, Shows final correction.

Fig. 7. Normal prominence of the upper lip. A, Profile of an average infant of 2 months of age. Note how far the upper lip protrudes beyond the lip and chin. B, Young infant with complete right sided cleft of the upper lip and palate, recently repaired over a wide open bony cleft without any attempt immediately to replace the premaxilla, thus retaining approximately the normal relationship of the upper lip to the lower lip and chin for this age.

Fig. 7A.

Fig. 7B.

Fig. 8. Normal prominence of the upper lip. A, Shows the prominence of the mid portion of the lip in a 12 hour old baby with a complete left sided cleft of the lip and palate. B, Shows this prominence persisting 12 days after the repair of the lip over the open bony cleft. C, Note the amount of retraction of the upper lip that has occurred during 9 months' subsequent development. If, at the primary operation on a young infant, the upper lip is not given its natural prominence at this age, in subsequent growth the upper lip is apt to become very much retracted.

Fig. 8A.

Fig. 8B.

Fig. 8C.

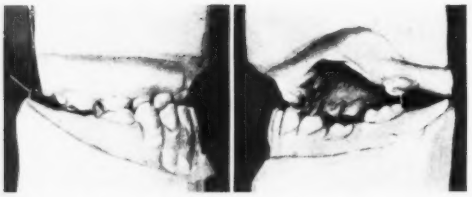


Fig. 9. Maxillary wiring, tooth destruction. Shows an average amount of retraction of the upper jaw following early forceful closure of the cleft by bone wiring, but also note the loss of the teeth on the cleft side with probable destruction of the germs of the corresponding permanent teeth.

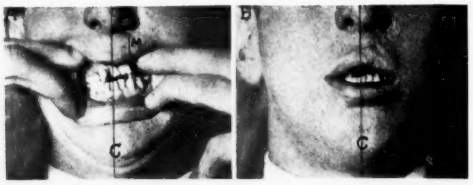
3. The premature loss of the first, and subsequent derangement or a possible loss of second, teeth following the transfixing of the jaw may be the source of expense, disability, or even untimely death in the producing period of life (Fig. 9).

4. Transfixing the jaw with wires is an unnecessary step and one that does not of itself accomplish the object for which it is advocated, namely: the better ultimate adjustment of the lip and nose (Fig. 10).

Though the illustrated records of the earlier operators are very meager, the descriptions they have left give us reason to believe that certain surgeons doing this work in the first part of the last century had developed great skill in the planning and cutting. The results must have been compromised, however, by the use of the "harelip pins" which were dictated by the lack of anæsthesia. We can today accomplish by proper undermining and careful suture of the soft parts

A. B.

Fig. 11. Repair of lip over open alveolar cleft. A, Shows the characteristic external deformity of the soft parts in the presence of a complete single cleft of the lip and palate. B, Shows the replacement that can be made over the wide open bony cleft. Note that the nostrils, columella, and tip of the nose are approximately symmetrical to the midline of the face. See Figures 12, 13, 14, and 15.



A. B.

Fig. 10. Maxillary wiring, failure of accomplishment. A, The midline of the premaxilla indicated by the line *M* has been brought past the midline of the face *C* in a complete left sided cleft by a bone wiring operation done in early infancy. B, Note that in spite of this overcorrection of the original maxillo-pre-maxillary displacement, the base of the columella of the nose remains drawn over to the right. This is just one demonstration of the fact that the distortion of the nose in a single one sided cleft of the lip is but little dependent upon the interrelationship of the underlying bones. See also Figures 11, 12, and 13.

everything that is claimed for the direct bone adjustment, and, on the average, can do better work for lack of distraction of the attention from the essential points (Fig. 11).

LATERAL DISPLACEMENT OF NOSE, SPREAD NOSTRIL

In a complete cleft of the lip and palate, the lateral deviation of the nose is, no doubt, due partly to the maxillary, and, with it the premaxillary, displacement, but uniformly there will be almost as much nasal displacement with a complete cleft of the lip and an intact alveolus (Fig. 12).

The nasal deviation is due mostly to an actual change in the relation of the soft tissue and cartilages to the bone and comparatively little to the bony displacement (Fig. 12). Therefore, it is not to be expected that shifting of the bone by itself will restore either the soft tissues or the cartilages to their proper position (Fig. 10). On the other hand, with sufficient undermining, these soft tissues can be shifted far enough to compensate for both the soft tissue and the bony displacement, but this undermining must be almost as extensive on the non-cleft side as on the cleft side (Figs. 14 and 15). This is a story of "To have and" also "to hold," and unless certain adjustments are made and maintained, the nose will continue to deviate with subsequent growth.

To maintain the position of the bridge during the growing period, it is essential not only to bring the nose approximately to the midline but also to restore the normal relation of the labial end of the columella to the labial end of the ala. To accomplish this latter, the nostrils must be symmetrical, for the tip of the nose is composed of

Fig. 12.

Fig. 13A.

Fig. 13B.

Fig. 14A.

Fig. 14C.

Fig. 14B.

Fig. 15A.

Fig. 15C.

Fig. 15B.

Fig. 12. Nasal displacement. This cleft of the lip over an intact alveolus shows the characteristic spreading of the nostril and the displacement of the nose and columella to the opposite side.

Fig. 13. Nasal displacement. A, Shows little more displacement of the nose in the presence of a wide open cleft than was shown in Figure 12, where the alveolus was intact. B, Shows repair of the lip and restoration of the nose to the midline without changing the relation of the sides of the bony cleft.

Fig. 14. Unilateral undermining. A, Shows the displacement in a typical partial cleft of the lip with no bony cleft. Note that the transverse slit of the mouth is about symmetrical to the midline of the face but that the cartilaginous part of the nose has drifted somewhat to the right, that is, away from the lip cleft. The dotted line in the cheek in the drawing indicates the extent of the undermining that was done in this case which was operated upon some years ago. B, In this picture, the immediate postoperative result, it will be seen by the vertical lines that the mal-relationship of the lower part of the nose to the mouth was not corrected by this operation.

Fig. 15. Bilateral undermining. A, Shows a child with a complete right sided cleft of the lip and an open cleft in the alveolar process, with somewhat more nasal distortion than was present in Figure 14. The dotted lines in the drawing show the amount of undermining in this, a more recent case. In B it will be seen that with bilateral undermining of the soft tissues, the nose has been brought into proper relationship with the mouth and to the midline.

Fig. 16A. Fig. 16B. Fig. 16C.

Fig. 17A. Fig. 17B. Fig. 17C.

Fig. 18A. Fig. 18B. Fig. 18C.

Fig. 16. Obliquity of the nose. These 3 young infants show the varying amounts of obliquity of the nose which, in each, is somewhat proportionate to the width of the cleft. See Figure 17.

Fig. 17. Obliquity of the nose. Shows pictures of older children with about the same width of cleft as the 3 babies in Figure 16, and from this it will be seen that growth by itself, in the presence of an uncorrected cleft, has no tendency to correct the obliquity of the nose.

Fig. 18. Obliquity of the nose. Shows a young baby with a fairly wide cleft and with typical obliquity of the nose, which latter is almost completely corrected by the operation that closed the cleft. Figure B taken 12 days after operation. C, Shows the condition a year later in which the new position of the nose has been well maintained. Note that in both B and C there is a slight droop of the inner end of the ala on the cleft side not corrected by the operation, but this defect has no tendency to compromise the position of the nose as a whole.

the walls of the nostrils. Simply narrowing the transverse diameter of the floor of the vestibule without restoring its vertical height might, as far as we know, maintain the central position of the bridge but will not correct the lateral flattening (Fig. 22). These are very positive statements dealing with a very complex matter, but, over a long period of time, we have made a great many observations that lead us to believe that these statements are correct in the majority of cases.

Fig. 19A.

Fig. 19B.

Fig. 21A.

Fig. 21B.

Fig. 19. A, Shows a 16 year old boy with a complete cleft of the lip and palate, which had not been operated upon. The pencil line shows the midline of the face from which it will be seen that the nasal deviation also involves the bony bridge. B, Shows the condition about 3 months later. Comparing the nose with the penciled midline in this picture, it will be seen that the position of the bony bridge was not corrected by the operation and that the stiffness of the deviated cartilaginous septum has prevented the base of the columella from being brought to a symmetrical position. From now on it is probable that with any further growth of the face these deviations will decrease rather than increase.

Fig. 20. Obliquity of the nose. A, Shows a boy approximately the same age as the one shown in Figure 19, but on whom an average closure of the cleft had been done

Fig. 20A.

Fig. 20B.

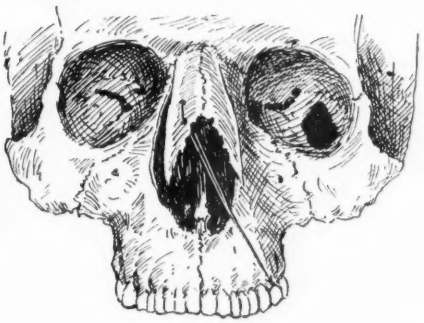


Fig. 20C.

in early infancy. As is very commonly the case, the proper relationship of the point of the ala to the base of the columella was not restored at this operation and, therefore, the deviation of the nose continued until it involved the bone as well as the soft parts. In this instance, the correction obtainable by readjusting the soft tissues would not have given complete satisfaction—therefore, the nasal bones and the lower end of the septum were mobilized and held in position by wires passed through the tissues and attached to the teeth. The drawing is taken from "Surgery and Diseases of the Mouth and Jaws," page 239. B, Shows the final result in which the position of the nose, as can be seen in the photograph, is not quite symmetrical, but on the patient this is hardly discernible.

Fig. 21. Obliquity of the nose. A, Shows flattening of the nostril which ordinarily occurs in the single partial cleft of the lip. For some reason in repairing this case instead of rotating the nostril into proper relation with its fellow, an excess was removed from the floor which gave the small nostril shown in B.

Of one thing, however, we are even more sure: there is no 100 per cent perfect about any of it (Figs. 16, 17, 18, 19, and 20).

In a single cleft of the lip, the long axis of the nostril on the cleft side is more transverse than that of its fellow, the nostril as a whole is somewhat posterior to its fellow, and the nose is correspondingly flattened on that side (Figs. 16 and 17).

To correct these nostril distortions, it is necessary, first, to mobilize all mal-related structures with the least amount of external scar; second, to draw them into the most natural form and position attainable; third, to fix them by suture, until healing has occurred.

TECHNIQUE OF OPERATION

Of a number of different plans that have been described for correcting the harelip, a few have gained wide acceptance. Each provides for the repair of an open cleft in the floor of the nostril but none that we know of goes far enough to give the best attainable nasal adjustment. This does not imply that our predecessors did not know

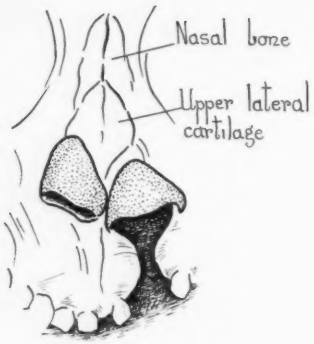


Fig. 22A.

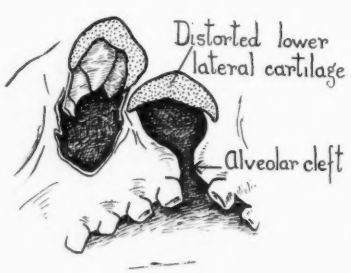


Fig. 22B.

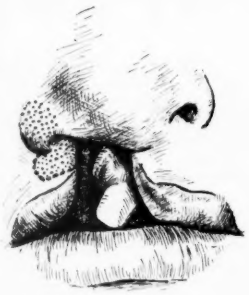


Fig. 24.

Fig. 23.

Fig. 22. Nasal distortion. These two figures are an attempt to semidiagrammatically illustrate the changes in shape and position of the lower lateral cartilage in a complete left-sided cleft of the lip and palate. There is always some distortion of this cartilage in almost every lip cleft; it is greater in proportion to the extent and width of the cleft (see Figs. 16 and 17) and becomes accentuated with growth in the uncorrected case. The outer surface of each cartilage is here shown in stipple, the cleft in the bone and the nasal fossæ are shown black. Note in A that the damaged cartilage has slumped caudally, more so in its median than its outer part. In B the lateral spreading of the cartilage as it spans the cleft is more clearly shown. As the skin of the tip and ala is closely adherent to the cartilage that side of the tip and ala will be correspondingly flattened. No attempt to restore the contour of the nose will be successful that does not correct the distortion and displacement of this lower lateral cartilage.

Fig. 23. Tissue mobilization. The line of dashes indicate the mucous incisions through which the undermining is done that frees the cheek and lip tissues from the bone in a plane superficial to the periosteum. The heavy dotted line across the nose indicates an incision in the lining of the vestibule and through or above the lower lateral cartilage from which an undermining plane extends between the external skin and the lower lateral cartilage as far down as the reflexion at the free border of the ala. The coarse stippling indicates the extent of the undermining between the soft tissues and the bone. The fine stippling indicates the area of undermining between the outer surface of the lower lateral cartilage and the skin of the nose. The downward turn at the outer end of each fornix incision gives greater relaxation of the lip and is made just in front of the parotid duct opening. Along the fornix the incision is made far enough away from the bone so as to leave a sufficient full edge of mucosa to which to suture if desired.

Fig. 24. Thinning the spread ala. The stippling indicates an area from which is removed, subcutaneously, a wedge of the excess tissue that occurs between the lining and covering in the outer part of the flattened ala. Above and internal to the lip cuts indicated by dashed line, only the subcutaneous tissue needed to nourish the skin flap is retained. These steps greatly facilitate the subsequent molding of the nostril and the lip flap supplements the floor of the vestibule. (See Figure 56.)

how to do this, but we could find no description of a systematic plan recorded. We have, therefore, been led to work out certain definite plans for dealing with tissues surrounding the deformed nostril which might be used in connection with any one of the accepted plans of lip repair.

The first step is to mark off on the lip the plan of the external skin incisions. This will outline the raw surfaces to be united by sutures. Upon the accuracy of these cuts will depend the possibilities of the ultimate result. Therefore, while they are first planned and measured off with the eye, they are checked up with fine pointed dividers and pricked in with aqueous methylene blue solution on a fine "crow quill" pen or a hypodermic needle, before any cuts are made, so that the landmarks are not obscured by the undermining and the accompanying flow of blood (Fig. 37).

After the lip incisions are outlined, it is well again to identify the pertinent points in the nasal distortion with which we are about to deal. (Compare with Figure 22 Figs. 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 26, 27, 29, 30, 32, and 33, which show various types of nasal distortions.)

The first operative step is the mobilization by undermining of each distorted or displaced tissue. This should release the restraining tissues from their underlying bony attachments, and at this stage cartilage may have to be shifted in its relation to bone, its fellows, or the overlying skin (Fig. 23). If necessary, excision of excess tissue between the lining and covering of the flattened ala may be made at this time (Fig. 24). The next

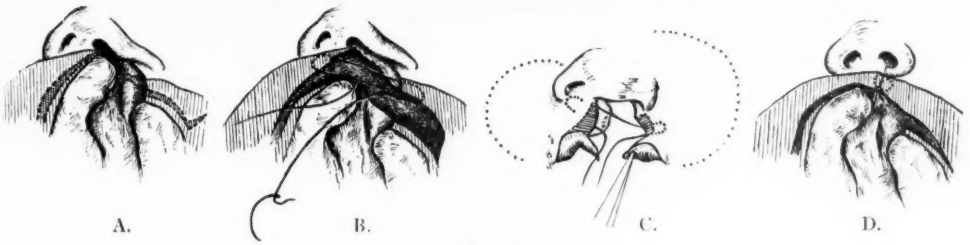


Fig. 25.

Fig. 26A. Fig. 26B.

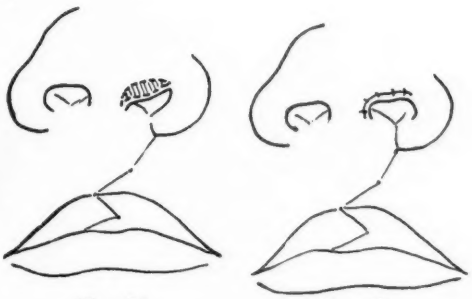


Fig. 28A. Fig. 28B.

Fig. 27A. Fig. 27B. Fig. 27C.

Fig. 25. Restoration of the floor of the nose. A, Shows palate and labial fornix viewed from below with lip and cheeks cut away. The "cross hatched" area shows the line of the mucous incisions and undermining planes. B, Shows the path of this suture viewed from below and the soft tissues being drawn forward from the bone. C, Indicates the course of the chromic gut suture viewed from in front. D, Shows the reposition of the tissues that results from the drawing up and fixation of this suture. This suture should not include any derma or it may cause some hesitancy in healing. *The proper placing of this suture is essential to the proper molding of a distorted nostril.*

Fig. 26. Nasal obstruction. A, Shows a complete cleft in which the ala is drawn out into a flat band, the lining wall being relatively the same length as the external skin covering. If this flat ala is tubed into a nostril, even after the lining is separated from the covering, the lower lateral cartilage and the skin lining will bend into and cause an obstruction in the nostril as shown in B. Packing the nostril with gauze and retaining the pack for some days after the operation will help correct this. Greater accuracy in distributing the lining is obtained by through-and-through mattress sutures (Fig. 27B). Formerly, we incised the mucosa and lower lateral cartilage to lessen this deformity.

Fig. 27. Nasal obstruction. A, Shows a baby with an alar distortion similar to that shown in Figure 26. Besides thorough undermining of the ala, the proper relation of the lining to the covering was obtained by 2 or 3 mattress sutures tied on the external surface, B. This, in addition to the gauze packing, gives a much more patent nostril.

Fig. 28. Nasal distortion. The procedures illustrated in Figures 23, 24, 25, 26, and 27 will restore the floor of the nostril, base of the columella, and the floor of the ala in all infants and young children but in most cases after these maneuvers have been completed, there is a downward droop of the inner half of the affected nostril (see Fig. 22A). In all but older children this can be corrected by removing a crescent of skin, A, from above this distorted part of the border and suturing the resulting defect as shown in B. Ordinarily, the excision goes down to, but not through, the underlying cartilage. If it has not already been done, this latter is thoroughly freed from the overlying skin by inserting a dissecting scissor into the opening before the defect is sutured. This suturing then draws the slumped cartilage up to its proper level. When indicated this crescent should be removed before the insertion of the mattress sutures shown in Figure 27, but the latter is necessary only if the outer half of the ala has been flattened out in a straight line.

Fig. 29A.

Fig. 29B.

Fig. 30A.

Fig. 30B.

Fig. 31A.

Fig. 31B.

Fig. 29. Nasal distortion. In young infants this crescentic removal may be necessary to correct a downward displacement of the cartilage shown in Figure 22A, but not the flattening shown in Figure 22B. In the case here shown, the B deformity shows very plainly but if viewed directly from the point, it may be seen that the A deformity is not present; therefore, it was not necessary to remove a crescent from above the inner half of the nostril in this infant.

Fig. 30. Nasal distortion. In this infant with only a partial cleft, the downward droop of the lower lateral cartilage shown in Figure 22A was quite pronounced; therefore, a crescent was removed.

Fig. 32.

Fig. 33A.

Fig. 33B.

Fig. 33C.

Fig. 33D.

Fig. 31. Nasal distortion. In this infant there is quite a deep notch on the uncleft side at the junction of the nostril border with the columella as seen from the front. Therefore, a crescent was taken out on the uncleft side to make the two symmetrical.

Fig. 32. Nasal distortion. In older children and adults the manipulation and suturing shown in Figures 25 and 28 may not restore the proper relation of the 2 halves of the columella, that is, it will not correct the backward displacement of the inner half of the lower lateral cartilage (Fig. 22B), and to do this it may be necessary to split the columella deeply through an external incision and substitute a triangular excision for the crescentic excision described in Figure 28.

Fig. 33. Nasal distortion. Shows a 16-year old girl with pronounced cartilage distortion operated upon by the plan shown in Figure 32. Viewed from the front, the border of the ala on the sound side presents a gentle concavity while on the cleft side it is of a very pronounced convexity. In this case there was very little real bony distortion of the nose and the whole nasal distortion was corrected by the operation on the soft tissues.

step in the actual operation is the making of at least the upper part of the incisions on either side of the cleft lip. For continuity of description, however, we will, for the present, assume that lip incisions have been made and proceed at once with the molding of the nostril.

The adjustment of the floor of the nose is made as shown in Figure 25, care being used to prevent

Fig. 34A.

Fig. 34B.

Fig. 36.

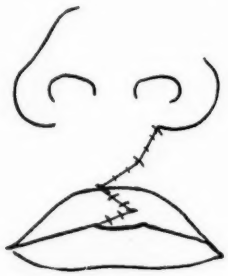
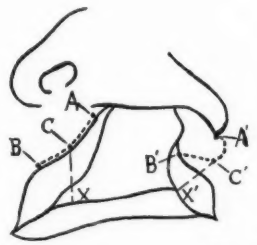


Fig. 35.



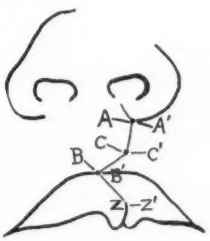


Fig. 37.

Fig. 34. Suture scars. In both these cases the greatest difficulty in the re-operation is the elimination of scars caused by badly placed sutures. In A the whole of the scarred area of the lip was excised which made an undesirable tightness from side to side. In B the two transverse scars were excised individually and the defects closed by fine skin sutures in the hope of making these less visible.

Fig. 35. Suture scars. Objectionable suture scars can be avoided by placing all stay sutures from the mucous surface and approximating the epithelium with fine sutures that engage not more than 1 millimeter on either side and are not pulled too tight. Horsehair is apt to make better scars than silk and the postoperative care of the wound may have something to do with the result.

Fig. 36. Mirault operation. Lip incision. We have had some difficulty in making out just what was Mirault's original operation. This picture is in Henry Hollingsworth Smith's *Operative Surgery* (1852), and is labeled "Mirault's operation." It was upon this that the following operation was based, but the details as given in this paper were gradually worked out from our own experience. One might infer from studying the figures that the correction of the spread nostril was not part of the operation. (The usual nasal deformity is not shown.) Note also that the raw surface corresponding to $A'C'$ in our Figure 37 is longer than the $C'B'$ surface. This will work out possibly more satisfactorily than our plan, in partial lip clefts, but for uniformity, we use the plan shown in Figure 37 in almost all cases of primary operation.

The statement has been lately emphasized that using a displaced flap in the repair of a lip cleft would cause muscular distortions in the movements in the new lip; with this point in view, we have recently made a study, both directly or by means of movie films, of about 30 cases operated on by this plan, and, in none of them, after the immediate postoperative stiffness disappeared, were there any asymmetrical contortions evident.

Fig. 37. Lip incision, Mirault operation. The points A, B, and C, and A', B', and C' are the ones to be pricked in before the undermining. A is placed on the mucocutaneous junction just above the point at which a line corresponding to the oblique base of the columella would intersect the vermilion. Usually in a complete cleft of the lip, there is a very slight shallow notch in the skin at this point; B is placed just where the ridge that bounds the philtrum on the opposite side meets the mucocutaneous junction; C is a point half way between A and B. While it was stated that the points A, B, and C were placed in the mucocutaneous line in practice, they are put just within the skin border so that when the incisions are made, the marks will still be visible as guides in placing the sutures. In a partial cleft, A is located just on the inside of the defect of the lip instead of along the mucocutaneous junction which in this instance does not extend up this far.

On the outer side of the cleft, A' is put just beyond the point of the ala (see Figures 38 and 42). By drawing the lip downward and outward, the exact point where the ala joins the lip will become visible. The placing of point C' requires some consideration. It should be under, and rather internal than external to A', and at a vertical distance from the vermilion border equal to CX (see Figures 43, 44, 45, 46, and 47). X is supposed to represent the future level of the vermilion border at this point; B' is on the mucocutaneous line at a distance from C' equal to BC. The distance from A' to C' must be equal to or less than the distance AC, but if $A'C'$ is less than the distance AC, the cut is brought to the proper length by making it curved as shown in an exaggerated fashion in this illustration.

In suturing the lip, A' is brought to A', C' to C, and B' to B, but before suturing B' to B, it is necessary to make the incisions BZ and $B'Z'$, the method of doing which is shown in Figure 48. Before finishing the vermilion border, the length of the skin part of the lip on either side, from the point of the ala to the mucocutaneous line, is checked up for symmetry, and, if there is a discrepancy in length, it should be corrected at this time (see Figures 42, 43, 44, 45, and 46). No cut is made from A' to B' at this time because some or all of the skin included within the area A', C', B' can be utilized in forming the floor of the vestibule, a place that is sometimes very deficient in lining.

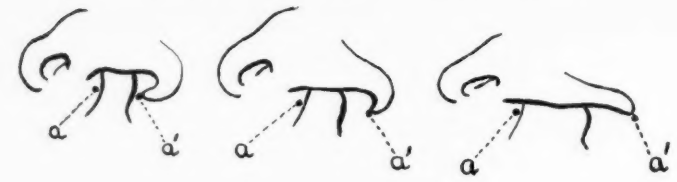


Fig. 38-I.

Fig. 38-II.

Fig. 38-III.

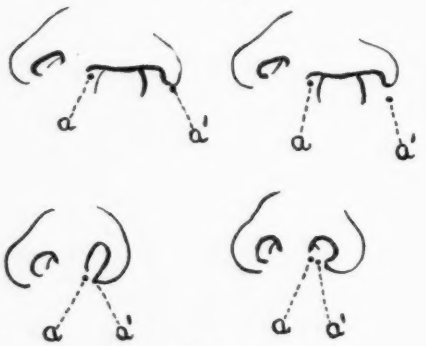


Fig. 39.

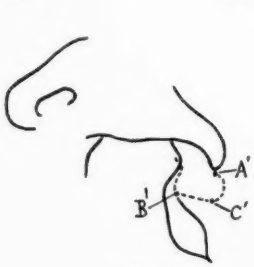


Fig. 40.

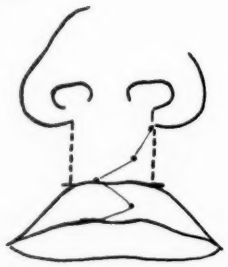


Fig. 41.

Fig. 38. Lip incision. Point of the ala. Normally, the edge of the border of the nostril, which is the border of the ala, meets the ala labial crease at a very acute angle that points almost directly toward the midline at the level of the junction of the columella with the lip. In a very slightly spread ala, the direction of this point is not much changed, the width of the floor being due mostly to the fact that the base of the columella has drifted over to the opposite side (see I in this Figure). In a moderately spread cleft, the direction of the point of the ala will be downward and somewhat inward (see II). In the young infant with a widespread complete cleft, the ala is drawn into a tight band across the cleft and the direction of the point of the ala is downward and outward as in III. If the A' is always put in the direction of the point of the ala, then when A' is brought into conjunction with A, the ala will be pointing toward the midline which is the right direction.

Fig. 39. Lip incision, floor of the nostril. Very rarely it apparently occurs that there is not enough tissue to form the floor of the nostril. More tissue for the floor can be gained by moving A' a few millimeters from the point of the ala but always in the direction of its axis. However, this will impinge on tissue that, as a rule, cannot be spared, and, as a result, the angle of the mouth on that side will be drawn toward the midline. (See Figure 47.)

Fig. 40. Lip incision. Vermilion border. Figure 49 shows a central notch in the vermilion which, in our earlier experience in this type of operation, was persistently annoying. It was finally overcome by not continuing the cut $C'B'$ directly through the vermilion border but from B' continuing upward along the dashed line shown in this Figure to include all of the available vermilion as part of this flap. The manner of using this extra piece of vermilion is shown in Figures 48 and 50.

Fig. 41. Vertical measurements. On completion of the approximation of the skin borders of the cleft, it is well to measure with calipers the vertical length of the lip on each side as indicated by the dashed lines in this figure. If these are unequal, the cause and correction should be considered at this time.

the distortion shown in Figure 26B. If necessary, the mattress sutures shown in Figure 27 are used. Before they are put in, however, look at the patient squarely in front to see that there is no downward droop of the inner half of the upper border of the nostril. If there is such a droop or if the inner half of the upper border of the nostril and the corresponding border of the columella make one straight line, then, in a young child, the crescentric excision (Fig. 28) is made. In an adult, the plan in Figure 32 is used (see also Figs. 29, 30, and 31). The steps shown in Figures 27 and 28 are carried in mind and if necessary are used and completed toward the end of the operation.

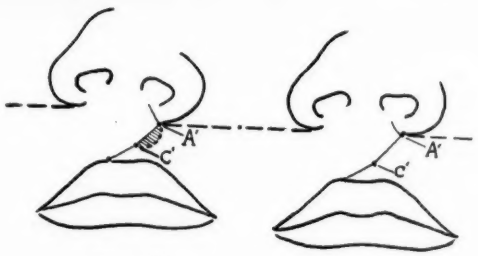


Fig. 42-I. Fig. 42-II.

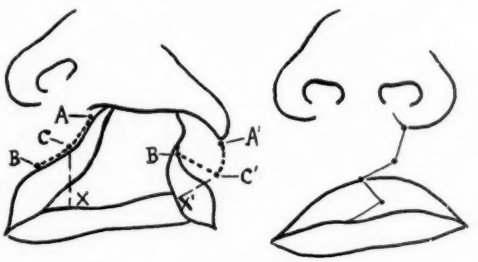


Fig. 43-I. Fig. 43-II.

Fig. 42. Lip incision. Locating A'. We take it for granted that the distance from the point of the ala to the mucocutaneous junction on the sound side of the lip is normal. If it is now found to be shorter on the cleft side than on the normal side, it may be that the point of the ala on the cleft side has been placed lower on that side than on the normal side (see I). By giving more concavity to the incision, $A'C'$, A' can be shifted up to its proper level (see II), but it may be necessary to replace the original buried chromic catgut suture (Fig. 25). It will be noted that when the discrepancy is due to a downward displacement of the point of the ala that it has not disturbed the symmetry of the border.

Fig. 43. Lip incision. Locating C'. If A' has been properly placed, and the distance from the ala to the cutaneous border is short on the cleft side, it will probably be found due to having placed C' too close to the mucocutaneous border, that is, $C'X'$ is shorter than CX, or $A'C'$ is shorter than AC; or both $A'C'$ and $C'X'$ may be short. Usually the angle of the mouth will also be found to be at a higher level than its fellow. For the correction of this, see Figure 44.

Fig. 44. Lip incision. Point C' continued. To correct the condition depicted in Figure 43, it is necessary to lengthen the incision $A'C'$ by making it more concave and then suturing C' further along toward B as shown (see II) and to compensate for this, either the flaps $C'B'Z'$ will have to be shortened as indicated in the dashed line in II or the incision CB will have to be extended toward the sound side. Shortening the flaps $C'B'Z'$ as shown in Figure 44 is the more apt to be the correct method.

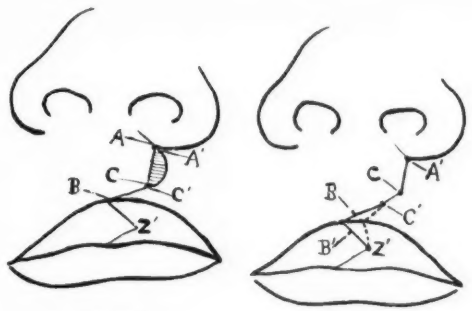


Fig. 44-I. Fig. 44-II.

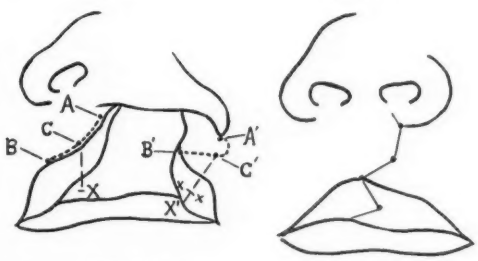


Fig. 45-I. Fig. 45-II.

Fig. 46-I. Fig. 46-II.

Fig. 45. Lip incision. Point C' continued. If A' has been put at the proper level, and the distance from the point of the ala to the skin border is longer on the cleft side than it should be, as shown in II, this may be due to having made $C'X'$ longer than CX, as shown in I (see Figure 46).

Fig. 46. Lip incision. Point C' continued. This condition is corrected by moving C' downward and outward as shown in I, and then uniting the point C'' to C'' to give the adjustment shown in II. The striped area shows amount excised in doing this.

The immediate proper adjustment of the lip itself is of secondary importance to that of the nostril. No matter how poor the adjustment of the lip in infants, it will of itself pull the separated halves of the alveolar cleft into a good relationship (Figs. 2 and 3). (In older children and adults with unyielding bone formation, the separation may remain.) But a poor adjustment of the nostril will be followed by increasing deformity of the bones and of the cartilages of the nose.

The most important point in the operation on the lip is not to excise too much tissue from the

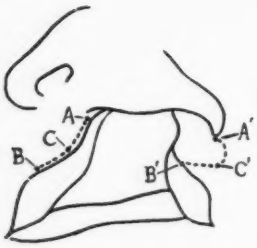


Fig. 47-I.

Fig. 47-II.

Fig. 48.

Fig. 49.

Fig. 50-I.

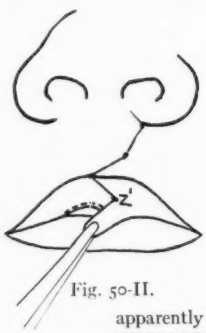


Fig. 50-II.

Fig. 50-III.

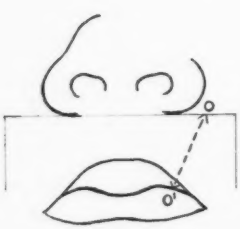


Fig. 51-I.

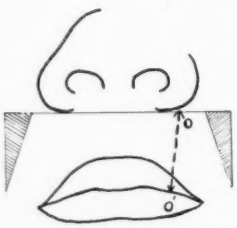


Fig. 51-II.

Fig. 47. Lip incision. Point C' continued. If the outer lip fragment is deficient in size, it will be found necessary to place the point C' a little external to the position of A', as shown in I of this figure, and, as a result, on the completion of the suturing, it will be found that the corner of the mouth on that side will be closer to the midline than its fellow. No attempt is made to correct this at the present. If it is in a young baby, there will probably be considerable spontaneous improvement with growth.

Fig. 48. Vermilion border. To locate the incisions $B'Z'$ the ends of the 2 vermilion flaps are grasped with forceps and drawn across each other as shown in Figure 48. But these flaps are put on very little tension, and the incisions are made a little closer to the distal end of the flap than apparently is necessary and the incision goes through the mucous membrane on the anterior surface of the flap only, the distal part not being entirely detached. (See Figure 50.)

Fig. 49. Vermilion border. The notch shown in the center of the lip was a persistent defect in an otherwise rather satisfactory operation until we learned to conserve all of the vermilion on the outer border of the cleft (see Figure 40).

Fig. 50. Vermilion border. I illustrates the defect that is apt to occur when the cut $C'B'$ (Figure 37) is carried straight across the vermilion. II shows the method of utilizing this extra bit of vermilion (see Figure 40) and III shows the flap in place. As a matter of practice we conserve the vermilion on both sides of the cleft and occasionally the notch is so situated that it is the flap from the Z side rather than the flap from Z' side that we finally use.

Fig. 51. Vermilion border. Not infrequently when the lip has been thus far completed, it will be observed that the visible vermilion border is too narrow on one or both sides as shown in I. By making an incision on the mucous surface corresponding to all or a part of the line shown in I on one or both sides as may be needed and suturing the mucous lining more toward the midline as shown in II, the hidden vermilion will be let down into view. The stripped areas are left raw to be later drawn together by scar. The vertical incision as shown is not always necessary; by running the transverse incision back far enough in the sulcus, the mucosa may be stretched.

lip or any at all from the lining of the nose. It should be borne in mind that in wide open clefts of the lip and floor of the nostril, part of the vestibular lining has slumped down until it appears to be on the surface of the lip, in which position it is in danger of mutilation unless the incisions have been properly planned. Removal of any of the lining causes permanent obstruction of the nostril. Removal of an excessive amount of tissue at the cleft borders will make a lip that is a little too long from above downward or one that is excessively long and flat and shortened from side to

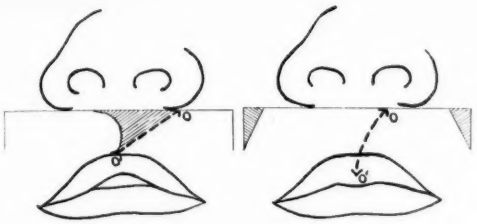


Fig. 52-I. Fig. 52-II.

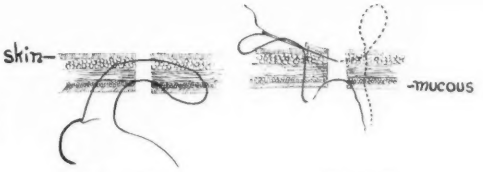


Fig. 53A. Fig. 53B.

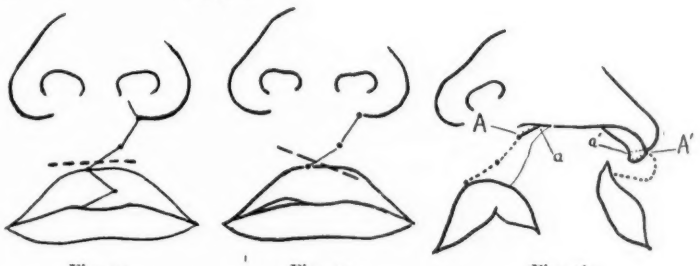


Fig. 54. Fig. 55. Fig. 56.

Fig. 52. Vermilion border. A certain irregularity in the vermilion border at or near the midline can be corrected as shown in this figure. For details, see legend under Figure 51.

Fig. 53. Retention sutures. The retention sutures in the lip are usually of horsehair and put in from the mucous surface in the form of a vertical mattress that preserves the full thickness of the lip; A shows the type of suture that may be used with a moderately loose lip; B shows the suture used by Lane. It is put in with a straight needle that punctures the skin on each side and re-enters through the same puncture hole. We find this useful particularly in the double harelip. In young babies there is apt to be a suppuration on the seventh or eighth day at the site of the skin puncture, due, probably, to having engaged some of the skin. It is annoying to have such a complication occur at this time and to avoid it we make a small stab wound through the skin at each point at which the needle is to emerge, and try to make sure that none of the derma is engaged in the loop.

Fig. 54. Retention sutures. In babies with single cleft, two of these retention sutures are used. The position of the lower one is illustrated in this figure. The second suture is put in above the *C* point. In adults, we sometimes use three sutures, one higher up.

Fig. 55. If there is a little extra fullness of the vermilion visible on one side or the other, it can be tucked up and permanently cared for with this suture, if the suture changes the relationship of the mucosa on the two sides.

Fig. 56. Diagram explanatory of the use of the skin flap *A'C'B'* in Figure 37 and Figure 24. The incision has been made and the flap is retracting up into the vestibule. After the suturing of the lip has been completed, an estimate is made of the amount of lining required for the floor and the lateral bulge of the vestibule. If there appears to be a real excess, it is trimmed along the dotted line *A'*, *a'*, and the resulting cut border is united along the free edge above *A*. The estimate of the amount of lining required should be very generous or the child may return later with a narrowing of the floor in the back part of the vestibule that was not evident after operation.

side and with an apparently protruding lower lip, the degree of deformity depending upon the amount of tissue that has been sacrificed. Bad suture scars are almost as great an evil (Figs. 34 and 35). Correction of these deformities immediately or later is difficult.

MIRAULT OPERATION[1]

We have had experience with three different operations consecutively. The Mirault type was taken up 10 years ago and has been used ever since (Figs. 36 and 37). The Rose[2] operation was finally abandoned on account of the difficulty in controlling the tendency of the reconstructed lip to be inartistically long. The Owen operation, which is a decadent form of the Mirault, was also abandoned because the results were still more objectionable. The logic of the Mirault plan is that a flap is taken from the upper part of the lip where there is excess tissue and implanted into the lower border where tissue is most needed. As the flap must of necessity carry vermilion border, this plan would apparently be the one best suited to partial clefts with only a slight notch. However, we prefer to use this method in all cases of single cleft rather than to risk vitiating the technique by working with different operations.

[1]Mirault. Operation for harelip. J. de chir., 1844, ii, 257. This is probably the first reference but the operation is illustrated more clearly in H. H. Smith's Operative Surgery, 1852.

[2]Surgery and Diseases of the Mouth and Jaw, p. 233.

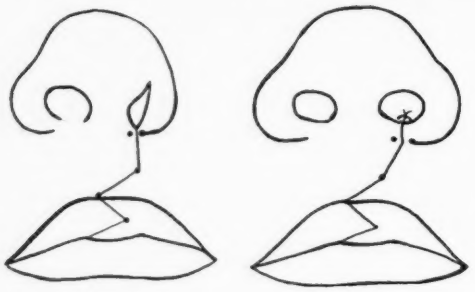


Fig. 57-I. Fig. 57-II.

Fig. 58.

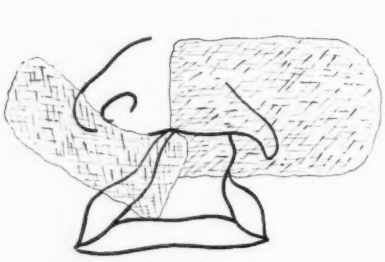


Fig. 59A.

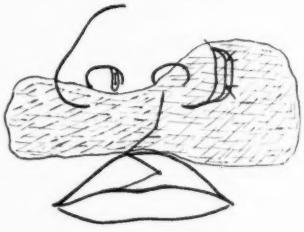


Fig. 59B.

Fig. 57. Floor of the nose, suture. The *A-A'* suture does not control the nostril directly and, in spite of the chromic gut suture shown in Figure 25, that part of the vestibule lining on either side of the cleft that should form the floor will drop downward into the palate cleft, giving somewhat the appearance shown in I of this figure. One or two sutures put in the floor of the nostril with a small heavy Lane palate needle as shown in II completes the tubing of the lining and corrects most of the difficulty. Theoretically, one could put in this nostril suture more easily before putting in the *A-A'* suture, but *A-A'* demands the greater accuracy, and, therefore, comes first. It is also our practice to delay the removal of the crescent of skin from the ala (Figure 28) until after the suturing of the floor of the nostril is completed. Only then can one tell accurately just how much of a removal is to be made (Figures 24 and 56).

Fig. 58. Nostril retention suture. To protect the union at the floor of the nose and the upper part of the lip during the first 12 days, the suture shown in this figure is used, usually of double horsehair. It loops over a flat lead plate (1 millimeter thick) in the sound nostril well up on the septum, and over a molded one in the ala labial fold. Before inserting this suture, the hæmostatic packing is removed, and before drawing the suture taut, the reconstructed nostril is lightly packed with gauze saturated with 5 per cent xeroform ointment made up in vaseline. This keeps the nostril distended to its proper size and prevents a leakage of serum from the nostril onto the suture line which latter is to be kept absolutely clean and free from clots.

Fig. 59. A, Hæmostatic packing. When the undermining incisions are made, no attempt is made to catch the bleeders, except possibly in adults, but folded gauze is inserted into the undermining planes and pressure is applied from the outside until the active bleeding is controlled. Unless the baby should be in bad condition, these packs are removed before the lead plate suture shown in Figure 58 is inserted, and are reapplied after this. The approximate position of these packs is shown during and after the operation.

In examining any case of complete single cleft of the lip in a young baby, the following points should be noted:

1. The ala is stretched into almost a straight band across the wide open bony cleft and the floor of the nostril is difficult to identify.

2. The base of the columella lies obliquely, lower on the cleft side.

3. The outer part of the lining of the vestibule has been displaced downward and inward.

4. The vertical distance between the point of the ala and the vermilion border on the cleft side may be shorter than on the opposite side and, therefore, the mouth slit has an obliquity which is the reverse of that of the columella.

5. Though it may not be in evidence, usually enough tissue to make the nostril floor will be found lying below the base of the columella on the cleft side. This is usually supplemented by using all or part of the small triangle between lines of incision on outer side of cleft (Figs. 24 and 37).

These are all important features in the topography of the field upon which the ground plan of the new lip is to be laid out.

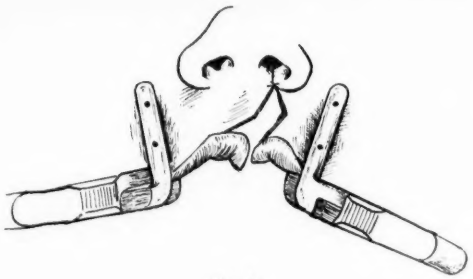


Fig. 60.

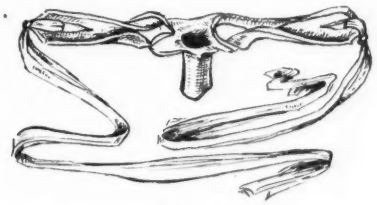


Fig. 62.

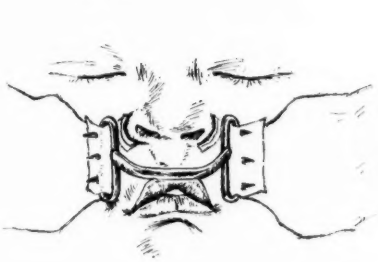


Fig. 61A.

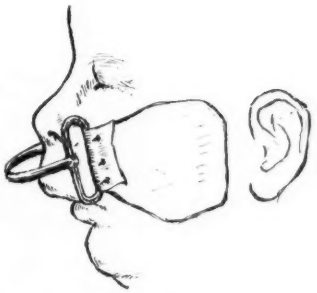


Fig. 61B.

Fig. 60. Hæmostatic clamps. Shows two angled bulldog clamps on which, to prevent them from slipping, teeth have been placed on the outer blade and two corresponding holes drilled in the inner blade. They remain in place while the skin is being sutured. The teeth puncture only the skin.

Fig. 61. External support, the Logan clamp. Some form of external support is desirable to supplement the sutures. Many plans have been devised for doing this but the only satisfactory one which we have encountered is illustrated. The point that gives it its pre-eminence is that Dr. Logan made it in the form of a buckle by which the slack which comes from the slipping of the adhesive plaster can be taken up as it occurs. These are put on within an hour or so after the operation.

Fig. 62. Breathing tube. Not infrequently the baby will have difficulty in breathing for some days after the reconstruction of the lip. The nostril that is not obstructed by packing is apt to be insufficient, or become obstructed by mucus, and the breathing may be obstructed either by the tongue or lips. This is quite detrimental to the baby. It is restless and loses weight. If such a baby is observed closely, it will be seen that there is some obstruction to the respiration at the mouth. If a soft rubber tube is passed into the mouth just to the oral pharynx and fastened in place, the baby will breathe comfortably, and the food can be administered with a dropper through the tube or along side of it. The tube can be held by a suture attached to a Logan clamp, or a longer tube can be used which is split as shown in the illustration, and fastened to the cheek with adhesive plaster or around the back of the neck with tape. The tube is retained as long as necessary but is taken out of the baby's mouth and boiled at least once every 24 hours. If it is too long, it makes the baby gag.

There is more apt to be an excess than a lack of tissue in the upper part of the lip, and, in most cases, some deficiency in the lower part of the cleft. The Mirault operation has been illustrated with the flap turned from either border of the cleft, but it is our practice always to turn the flap from the cleft side as it is easier to give symmetry to the vermilion border when the line of union is at the midline. It seems impossible, by an acceptable means, to restore the philtrum, but the lack of one bordering ridge is not very noticeable if the restoration is otherwise pleasing.

The plan of freshening shown in the illustrations usually gives a pleasing lip, but every detail is of importance; especially so is the adjusting of the mucous lining which gives the final little forward protrusion of the central part of the vermilion border that is so characteristically "baby." If the triangular piece of the lip bounded by $A'C'B'$ is completely removed, there is a tendency to a narrowing of the floor of the vestibule. This has led to the practice of making the cuts $A'C'$, and $C'B'$, and suturing the skin without detaching the resultant triangular skin flap from the vestibular floor. Later, when the floor is sutured, as much or as little of this flap can be retained as is necessary to insure ample width of the floor of the nostril.

PRE-OPERATIVE CARE

The cleft lip is corrected (over the open alveolar cleft, if such a cleft is present), as soon as possible after birth. During the first few days of life there probably remains some of the immunity to surgical shock which is necessarily present during the process of birth. Operation may be done in the first 24 hours. In our series no deaths have occurred from operations on 24 hour old babies. During the period of jaundice, usually from the fourth to the tenth day, the clotting time may be prolonged and operation is not done in this period. The technique of the operation at this early age is difficult but the advantages to the baby and its mother outweigh the disadvantage to the surgeon.

Thorough physical examinations, as for all surgical patients, are necessary. Active skin infections, respiratory infections—real or suspected—and prolonged clotting or bleeding times are contra-indications for operation. The patient having been accepted for operation, feeding should be allowed to within 4 to 6 hours and water to within 2 hours of operation. A final check on the physical condition should be made just before operation. A blood donor should be available.

POSTOPERATIVE CARE

Immediately after the operation, the baby is put in the care of a special nurse and is kept in the operating room until entirely awake from the anæsthetic and it is certain that the airway is open, that bleeding has ceased, and that shock is not present. Tap water is given by rectum to all patients, and, if necessary, saline under the skin. If much blood has been lost or shock is present or suspected in small infants, 20 cubic centimeters of the parent's whole uncitrated blood (without necessity of matching) is given under each pectoral muscle, and the areas are massaged until no lumps can be felt. If the depressed state persists, an intravenous transfusion is done.

The patient is placed on his abdomen to allow blood to run out of the mouth. The continuance of hæmorrhage is closely watched for by the nurse and surgeon. A catgut stitch, which is put through the tongue at the start of the operation, is left in and gives a good sure way of maintaining control of the tongue, and, therefore, the airway, while the patient is waking. This stitch is left in, in infants and children, throughout the first night.

Immediate attention is given the lip. It is kept clean of blood and mucus by almost constant gentle wiping with small gauze squares soaked in a solution of equal parts of alcohol and boric solution. This is especially important during the first few hours, as the blood serum that oozes out at this time, if allowed to remain, will make a very hard crust over the suture line.

When fully awake and in satisfactory condition, the patient is returned to the ward. Airways must be kept open, and possible hæmorrhage must still be watched for. Trained nurses and resident physicians are necessary for continued success in caring for these patients. Breathing tubes are frequently used (Fig. 62).

If the lip has been kept clean during the first few hours, the after-care while simplified must still be thorough. If crusts form, they may be loosened by wet packs or cold cream and then gently separated from the stitches. If there is superficial cellulitis or infection around the suture line, wet packs should be kept on most of the time. The quartz light is frequently used, both locally and generally.

For the first 24 to 48 hours, feeding is done with a sterile syringe with rubber tip, or with a spoon. After this, the baby may nurse the breast or use the nipple. Water by mouth is given as soon as the child will take it, and feedings are begun the same day. (In repair of palates, nipples are withheld for 2 weeks after operation.) The patient's hands must at all times be kept away from the mouth. For infants, a special cuff of wooden tongue depressors sewed into cloth is tied around the arm. These may be incorporated in the sleeves of the under jacket. For older children (and adults if necessary) padded anterior wooden splints are applied to prevent flexion of forearm.

The packs under the lip are removed in 24 hours. The skin sutures are removed in 5 days and the deep sutures in 12 days.

FROM THE SURGICAL CLINIC, UNIVERSITY OF ZURICH

CHONDROMATOSIS OF THE JOINT CAPSULE

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Assistant in the Surgical Clinic, University of Zurich, Prof. Dr. Clairmont, Chief

CHONDROMATOSIS of the joint capsule, first described in the year 1900 by Reichel, is a condition about which little is known. In the English literature contributions to our knowledge have been made by Henderson and Jones. More recent articles on the subject may be found in the German surgical literature. Of late the disease has been recognized more often because of this interest. For example, Forssell's X-ray plates showed the disease but he did not so name it. The X-ray plate is still the only safe means of diagnosis. Therefore, more X-ray plates of joints must be made in ill defined joint disease. It is true that even an X-ray plate may be a source of error. Schinz, in his compendium, states: "The cartilage foci may not be discernible in the X-ray plate; they become visible only when they calcify and ossify at the center." Hesse describes a case in which chondromata were present but they were not apparent in the X-ray film. Even though visible as chondromatosis in the film, it may be described as osteochondromata. One should say that a chondromatosis of the joint capsule is not an ossification but a calcification. With these exceptions the X-ray picture is, and probably will remain, the decisive factor in diagnosing chondromatosis of the joint capsule.

In spite of the fact that chondromatosis of the joint capsule was described 30 years ago, many authors still are concerned principally with its etiology. They apparently have given up the theory of Reichel—the transformation, the result of an infectious process, of the synovial membrane into a cartilaginous tissue. At the present time two other theories occupy the foreground in the literature: the tumor theory and the theory of the origin on a metaplastic basis as a result of chronic irritation. The principal proponent of the first theory is Lexer with whom Janker, Jones, Henderson, Rostock, and others agree. The hypothesis of Mueller that chondromatosis of the joint capsule is a malignant formation or tumor may be refuted, as one could never be certain that an infiltrative growth was present. The advocates of the latter theory are, among others, Lotsch and Beckmann-Ivarsson.

As we are not in possession of material obtained at operation for the purpose of microscopic study, we cannot take a definite stand as to the question of etiology. However, we believe that the relatively great number of cases in our series not only justifies a report of them but we believe from our experience with them that we are in position to make a statement as to the etiology.

While there are still many acknowledged facts regarding the complex subject of chronic traumatic and non-traumatic lesions of the cartilages (such as osteochondritis dissecans, arthritis deformans), there are still differences of opinion among the different authors. Kappis states: "The arthritic formation of cartilages in the capsule becomes in part so overrich that, on macroscopic examination, one may gain the impression that the lesion is not an inflammatory process but rather a tumor formation." Haberler says that the genesis of chondromatosis is not on the basis of a formation of tumor tissue.

A consideration of the actual facts, however, permits us to say that there are graded transitions between osteochondritis dissecans on the one hand and arthritis deformans on the other and also that there are transitions between the latter and chondromatosis. We believe that chondromatosis of the joint capsule is a disease entity and would wish to apply this fact in a theoretical sense rather than in a clinical one. Therefore, in speaking of this disease, we would prefer to use the term "chondromatosis" with secondary arthritis deformans, in the same sense that Axhausen used the term.

In Case 6 we have a case of chondromatosis in a congenitally deformed elbow joint. We believe that in this case it would be logical to search for the etiological factor in a disturbance of development. Nevertheless we believe that this mode of origin should not be considered as the usual one even though such a theory might be applied in the majority of cases.

This leads us to the very important question of the influence of a traumatic factor in the origin of chondromatosis. Trauma, as an etiological factor, is naturally refuted by the adherents of the tumor theory, but it is accepted as a contributing factor at least by some authors (Boehm, Hesse). In our opinion even a single trauma might be sufficient to cause a joint, altered by chondromatosis, to

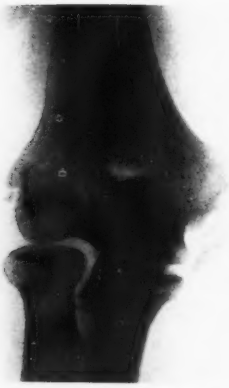

Fig. 1. Myositis ossificans after luxation of the elbow joint. The ossifications are not in the joint capsule but in the muscle insertions.

become painful and therefore unfit for function. On the other hand, we believe that chronic repetition of a trauma should not be rejected as a starting point in the etiology of chondromatosis of the joint. An example, therefore, may be seen in Case 9 in which chondromatosis occurred in an elbow joint which had been subjected to repeated trauma, the result of an early separation of a condyle of the humerus. It will hardly be necessary to show the great importance of this question and the many difficulties attendant upon establishing this fact. No doubt every case will have to be judged with the utmost care and as an absolute entity taking into consideration all possibilities of etiology.

The reason the clinical diagnosis is so extremely difficult is that the symptomatology of chondromatosis of the joint capsule may be hidden by the symptoms of some other chronic non-inflammable joint diseases or that the chondromatosis itself shows no characteristic symptoms at all. It would be useless to enumerate all of the possible symptoms which chondromatosis might present; no pathognomonic symptom will serve for all cases. But if one has in mind the possibility of chondromatosis of the joint capsule, he should be able to make, in classically advanced cases, a purely clinical, correct diagnosis; in all other cases, however, not to mention the already discussed rare exceptions without calcification, the X-ray picture will be the decisive factor in the diagnosis.

In the differential diagnosis first arthritis deformans should be ruled out and then osteochondritis dissecans. Clinically this will be almost impossible, even if the age and the history reveal certain hints. Only by means of the X-ray picture can one distinguish chondromatosis of the joint capsule from the other lesions already mentioned.

We wish to say that an X-ray picture of a fully developed chondromatosis of the joint capsule is so characteristic that there may be no reason at all to consider other joint lesions. The roentgenologist will always have only to distinguish the initial stages of this disease from other chronic joint lesions and the differential diagnosis between chondromatosis and osteochondritis will be established. In the latter the cartilage foci in the joint capsule will be missing; they are exclusively intra-articular or only ready to leave the "bed." In case of doubt this "bed" alone will establish the diagnosis between chondromatosis and osteochondritis dissecans, as the "bed" may resemble a defect in the cover of the cartilage of the relative joint surface.

The decision as to whether a chondromatosis of the joint capsule or an arthritis deformans is present, as has already been said, will usually depend on the X-ray picture. The characteristic variations of arthritis deformans on one hand—as pads on the edges, variation of the shape of the joint ends, defects of the cartilage, and reduction of the joint cleft—and on the other hand the characteristic cartilage islands in the capsule and in the joint in the presence of chondromatosis—with or without secondary arthritis deformans—are so unequivocal that there may be no doubt of the correct diagnosis. The picture is different in cases of arthritis deformans in which cartilage exuberance arises in the joint synovial villi. In such cases it will at times be difficult to be certain as to the diagnosis. All authors state there are really but few arthritic variations in chondromatosis of the joint capsule. Therefore, the relative number of the cartilage islands to the arthritic changes will make possible a definite diagnosis. As we believe there is a narrow boundary between chondromatosis of the joint and arthritis deformans, perhaps these borderline cases should be considered as merely transitional forms.

For the experienced observer of X-ray pictures, the differential diagnosis between chondromatosis of the joint and calcareous bursitis will not be difficult to make. In addition to the X-ray findings, the history will reveal the possibility of hæmophilic joints. The history, the shape, and the position of the calcified placques will easily establish the differential diagnosis between this and myositis ossificans. Trauma or luxation of a joint (particularly the elbow joint) will hardly

Fig. 2.

Fig. 3.

Figs. 2 and 3. Right elbow. Two small shadows are present near the head of the radius.

Fig. 4.

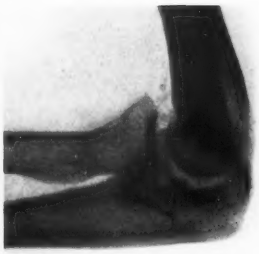

Fig. 5.

Figs. 4 and 5. The same case 1 year later. Calcifications in the joint capsule on the volar side in the radiohumeral joint. Typical chondromatosis.

ever be absent in the history of myositis ossificans. The ossifications may not be found so frequently in the joint capsule as in the muscle insertions and in the ligaments. The structure of the bones may easily be seen with the X-ray, and this is not the case in chondromatosis. Figure 1 shows the elbow of a young man of 18 years who had a classic myositis ossificans after luxation of the elbow. Bony deposits are seen especially in the lateral ligaments and show distinct bony structure.

Eleven cases of chondromatosis of the joint capsule were observed in the surgical clinic and the policlinic in Zurich. I was permitted to study 2 other cases which came from the physical institute for therapeutics (Professor Veraguth), and another case from Professor Clairmont's private practice. The X-ray pictures all belong to the scientific collection of the X-ray institute of the "Cantons" hospital of Zuerich and have been kindly loaned to me by Professor Schinz. I wish also to mention Dr. Friedl, assistant at the X-ray institute, who has helped to revise the X-ray pictures and has given me his opinion on them.

It is quite interesting to compare the X-ray pictures made at different times. Unfortunately, not all of our patients could be followed. We therefore have only one set of X-ray pictures of many of them. Some pictures have been omitted because they do not show anything in particular or because they are not suitable for reproduction.

Case 1. N.G., Policlinic No. 3414–28. Patient referred from the medical policlinic of Zurich with the diagnosis of chondromatosis of the joint capsule. In August, 1927, the patient had a paralysis of the right ulnar nerve, as a result of pressure. The paralysis disappeared entirely within a year. In August, 1927, by way of precaution, an X-ray picture of the right elbow was made. This showed near the head of the right radius two small shadows which could not be quite explained (Figs. 2 and 3). It was not until 1928 that the patient complained of limitation of motion when stretching his right elbow. Several X-ray pictures were made at different times. They showed (Figs. 4 and 5) a typical chondromatosis on the volar side in the radiohumeral joint. The number and the size of the chondromata grew.

The case shows nothing especially remarkable. It only emphasizes the importance of better follow-up of the patients from time to time if a center of calcification near a joint cannot be definitely diagnosed.

Case 2. M.E., aged 41 years, Policlinic No. 452–25. In 1909 he had an accident, injuring his right elbow in a machine. He was unfit for work for 3 to 4 weeks. The joint grew thicker but so slowly that the patient hardly noticed it. In 1925 an unexpected sudden movement while he was working produced a severe pain in the right elbow. After this accident he had limitation of motion up to about 110 degrees. Examination on February 5, 1925, showed a swelling of the entire right elbow joint. The swelling felt paste-like and granulous but was not fluctuating. It did not have the appearance of an inflammation. Extension amounted to 120 degrees, flexion to 75 degrees. The X-ray picture showed (Figs. 6 and 7) that the joint capsule was

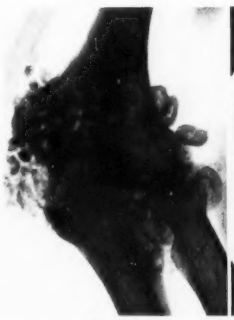

Fig. 6.

Fig. 7.

Figs. 6 and 7. The elbow is surrounded by a great number of round and oblong calcified knots. Near the radius head is a colony of "young" chondromata, only slightly calcified. The head of the radius is enlarged and osteophytes are present in the edges.

Fig. 8. Fig. 9.

Figs. 8 and 9. Calcified chondromata in the joint capsule situated only on the volar side of the radiohumeral joint.

interspersed with round and oblong calcified knots of groat and hazelnut size. The larger knots showed a light center and a dark peripheral shell. The joint cleft itself showed no calcified knots. Besides, there existed a marked arthritis deformans characterized by osteophytes of the edge of the enlarged head of the radius, or medial and lateral epicondyles of the humerus. The joint contours were uneven.

The history of this case showed two absolutely similar traumas. As a result of the first the lesion developed without symptoms; but as a result of the second the joint was made unfit for work. The growth of the chondromatosis islands by apposition is beautifully illustrated in the roentgenogram.

Case 3. H.W., aged 31 years, Policlinic No. 2027 28. Patient returned for examination to the surgical Policlinic early in November, 1928. In 1908 patient suffered a "trauma" with strong thickening of the right elbow. On February 10, 1928, he fell on the staircase, knocking his right elbow, which had previously been absolutely healthy. The joint swelled and became painful, and motion in the joint was limited. Seven days after injury an X-ray photograph was taken and showed a chondromatosis of the right elbow. (The pictures were kindly given to me by Dr. Ruppaner Samaden.) At that time he had burning pains in the joint, and extension was reduced by 15 degrees. Heavy friction was noticeable if the joint was forcibly moved. The X-ray photographs (Figs. 8 and 9) showed the typical picture of a purely volar chondromatosis with slight secondary arthritis deformans involvement. Comparison of these roentgenograms with the first X-ray pictures showed that a number of the calcified cartilage islands had increased in size. No chondromata were present in the joint cleft.

Fig. 12. Numerous oblong and round distinctly calcified bodies lie on the volar side of the radiohumeral joint. No signs of a secondary arthritis deformans.

Fig. 10. Fig. 11.

Figs. 10 and 11. Both joints (radiohumeral and humero-ulnar) surrounded and partly filled with a great number of strong calcified cartilage bodies arranged in the shape of grapes.

In this case as in the preceding, the patient had had two clearly marked traumas, the first having been received some time before the second.

Case 4. J. J., aged 34 years, Policlinic No. 621–24. Unfortunately, no details of the history of injury are available among the records of the chirurgical policlinic, and the patient did not answer our follow-up letter. However, the X-ray pictures show such an extensive and typical chondromatosis of the elbow joint that we are reproducing them (Figs. 10 and 11). Numerous calcareous cartilage shadows, arranged in the shape of grapes, are discernible. They partially surround the volar as well as the dorsal side of the joint, and lie between the radiohumeral and the humero-ulnar joint.

Case 5. B. E., aged 20 years, Policlinic No. 3184–21. Patient is supposed always to have been healthy. The present illness began 3 years ago, when he noticed pain in the right elbow when it was moved. The arm had never been injured. At first extension was restricted; he felt as if there was something too short in the joint. Later flexion was limited. There were times, however, when he was free of pain. When the weather changed the pain was aggravated. Three months before the examination the patient, who enjoyed strenuous athletics, began training by lifting and throwing. Thereafter the pain became so much worse that he came to the Policlinic. The X-ray photograph of the right elbow (Fig. 12) showed intact bones and very sharp joint contours. On the volar side of the distal end of the humerus were a great number of oblong and round bodies. These showed calcification in layers. Cartilage bodies were not definitely shown in the joint.

Fig. 13.

Fig. 14.

Figs. 13 and 14. Congenitally deformed right elbow joint. Subluxation of the radius with exostotic thickening of the head of the radius. Osteophytes present on edges.

Fig. 15.

Fig. 16.

Joint capsule contains numerous calcified chondromata.

Figs. 15 and 16. Same case as in Figures 13 and 14, but the chondromata are more densely calcified and are larger.

The patient did not appear in the follow-up as he was so busy. He wrote that, in his opinion, there had been no change in the condition of the joint, since the year 1921. We, therefore, believe that the condition must be a chondromatosis of the elbow joint which arose independently of a trauma. As the patient is a mason and continues steadily to do the heavy work of his trade, it would seem that the condition of the joint has either remained absolutely stationary or that the development of the chondromata of the capsule has progressed very slowly. The patient is entirely fit for work.

Case 6. H. A., aged 27 years, Policlinic No. 567–24. The patient stated that since he was 18 years old there has been a certain limitation of motion in both elbows. Early in 1924 the patient had pains in the elbows for 4 weeks. Following this there was limitation of extension. Since then there has been no recurrence of the pains. What troubles the patient most is that he cannot move the joints at once after they have remained for some time in the same position. He therefore has to move the joints slowly at first. For ordinary work the patient has no trouble at all. Both elbows present an abnormal projection of the head of the radius toward the radial side. When passive and quick movements are made the examiner has the sensation on volar side of the joint of a free body jumping off between his fingers. During such movement the patient suffers pain. Active motion causes no pain. At no time is there an appearance of squeezing in. Strong grating is noticeable in both joints, but is more marked on the left than on the right. Pronation on the left side is impossible and on the right side is very limited. The left arm is continually kept in a fixed middle position. Flexion on both sides is possible up to 60 degrees, extension up to 130 degrees. The X-ray pictures of both elbows are absolutely identical. Figures 13 and 14 are photographs of the right elbow. Pea and hazelnut sized calcified capsule chondromata are seen in the capsule of the radiohumeral and the humero-ulnar joint. Besides, there is a congenital anomaly, a subluxation of the radius with exostotic thickening of the head of the radius. The head of the humerus is not fully developed but is in only a rudimentary stage. Large osteophytes are present on the edges, a sign of a marked arthritis deformans. As compared with pictures taken in 1924, Figures 15 and 16 taken in 1928 show a distinct increase in the capsule chondromata.

The diagnosis would, therefore, seem to be a chondromatosis of the joint capsule in a congenitally deformed joint. As already stated, we attribute the lesions in both elbows of this patient to co-ordinated disturbances of development. It is remarkable that the patient can correctly carry on his work as a mechanician in spite of the high grade deformity of both elbow joints.

Case 7. K. J., aged 15 years, patient of Prof. Clairmont. This youth stated that he was hindered in extending his right elbow while playing tennis. He complained of no other difficulties. Examination of both elbow joints showed nothing remarkable except the limitation of extension mentioned. To determine whether a similar lesion was present on the left side, X-ray pictures of both elbow joints were made. Figures 17 and 18 are photographs of the right

Fig. 17.

Fig. 18.

Figs. 17 and 18. Right elbow. One round shadow is seen on the volar side of the humero-ulnar joint. The form and construction are typical for capsule chondroma.

Fig. 19.

Fig. 20.

Fig. 21.

Fig. 19. Severing of epiphysis of the epicondyle medium humeri.

Figs. 20 and 21. The same joint as Figure 19, 9 years later. Calcified chondroms in the capsule of the humero-ulnar joint. Great osteophytes of the edge on the radius head. Osteophytic layers on the place of the internal epicondyle of the humerus.

elbow. A round lime shadow of stratification is present on the volar side of the radiohumeral joint. The roentgenologist suggested a probable diagnosis of movable foreign body. The patient wished to be operated on, so on September 1, 1928, an arthrotomy of the right radiohumeral joint was done. Together with the synovia two very small, strong, gray knots came out when the joint was opened but no movable body could be found in the joint. However, the body, seen in the X-ray photograph on the volar joint capsule insertion of the humerus, was found to extend into the joint as a cherry-stone sized prominence; which was excised. With the probe the eminentia capitata felt somewhat softer than the other cartilage.

Roentgenograms of the left elbow also showed calcified cartilage islands of somewhat smaller size within the capsule of the radiohumeral joint.

In this case the diagnosis would seem to be chondromatosis of the joint capsule in both elbows in spite of the fact that the patient was a young boy of only 15 years of age. The history of a trauma could not be found. The softness of the cartilage on the eminentia capitata may be considered as the very beginning of an arthritis deformans. Because the patient went abroad a follow-up examination could not be obtained.

Case 8. S. G., Policlinic No. 552–25. In July, 1916, patient slipped in a stable and apparently fractured the right arm near the elbow. He was treated at the surgical clinic. The X-ray picture taken in 1916 (Fig. 19) showed no fracture but an old separation of the epiphysis of internal epicondyle of the humerus. The patient afterward worked without difficulty. Later, however, he suffered increasing pain and limitation of motion in the right elbow, which caused him to visit the clinic. The epicondyles were sensitive to pressure. At the side of the lateral epicondyle of the humerus down in the pit toward the olecranon was a free body which could easily be moved. Extension was possible to 130 degrees, flexion to 80 degrees. Extreme pronation and supination were painful. The X-ray pictures made in 1925 (Figs. 20 and 21) showed typical chondromatosis involving principally the capsule of the humero-ulnar joint. In addition enormous changes resembling arthritis deformans—osteophytes on the head of the radius—were present on the ulna and on both epicondyles. Osteophytic layers were seen on the medial side of the metaphysis of the humerus. The joint contours were uneven, the joint cleft moderately narrowed.

The enormous variation in this elbow joint must be due to a trauma (separation of the epiphysis) which was not recognized although it probably had been present since earliest youth. In all probability the chondromatosis and arthritis deformans may not be considered as the origin and cause of the difficulty but as co-ordinated factors in the production of the chronic irritated state of this joint.

Case 9. Policlinic No. 3187–28. Patient was 60 years of age. The history at the time he entered the surgical policlinic revealed that patient had had much pain. Since early in 1926 he had had pain in the right knee which was especially noticeable when he arose from a sitting position and while he was standing on his feet and as well when he mounted stairs; but he had no pain when he walked on level ground or when he sat still. Patient had consulted no less than 7 or 8 doctors. He had received all sorts of treatment including the violet and X-rays, and he had taken all kinds of medicine and household remedies but had not been relieved. With the exception of very slight thickening, the right knee showed no pathological conditions. However, flexion and extension were greatly restricted and caused pain. The joint was also sensitive to pressure to the side of the patella where the patient stated he was injured years ago. The X-ray photographs (Figs. 22 and 23) show calcified chondromatous knots varying in size from that of

a pea to a bean on the back of the right knee joint. The chondromata involved the joint cleft. No osteophytes were present on the edges, the joint contours were rough. Calcified chondromata were present in the metaphysis of the right femur.

The presence of chondromatosis of the joint capsule and at the same time bone-chondromata would indicate a congenital genesis. Whether the pain and the limitation of motion of the right knee had been due only to the chondromata of the capsule, which really were not so large, or whether the pain had been caused by arthritis deformans in the right hip, cannot be definitely stated. But as the left hip and the left knee were absolutely normal it is very probable that the chondromatosis was the original lesion and the arthritis deformans of the hip was produced only by the inactivity of the knee.

CASE 10. G. E., aged 42 years, female, teacher. The patient had had pains in her knees for a long time. She was not quite sure whether the left or the right knee gave the most trouble. Her knees frequently gave way and she felt very unsafe. She could give no reason for the affection. In the year 1926 she had had a course of medical treatment and her condition rapidly became better after a course of sea baths. She evidently never did have strong knee joints. In August, 1928, the pain in the right knee returned, probably caused by a sudden movement. This joint since then has shown the typical squeezed in appearance. The knee was swollen, the contours somewhat deformed, especially the upper part being bent upward and to the side. The patella was movable, the knee could not be entirely extended but stopped at about 165 degrees. Extension was painful. There were no abnormal movements, and no free body could be palpated. Circumference 34.5 centimeters. With all movements there was a marked cracking sound. Examination of the left knee showed no effusion, normal mobility, heavy cracking when moving, and circumference 33 centimeters. The anteroposterior X-ray photographs of both knee joints and the lateral X-ray picture of the right knee joint (Figs. 24 and 25) show pea-sized and bean-sized calcified cartilage shadows, especially on the front side of both knee joints. Arthritis deformans was also present in both knees, the lesion being more pronounced on the left than on the right side. Roentgenograms made in 1928 showed that the lime deposits had increased and that the layer of the calcified bodies had changed. Free joint bodies had already appeared on the right side and this would explain the appearance of the joint.

This was a case of typical chondromatosis of the joint capsule of both knee joints for which no satisfactory cause could be found. The fact that the lesion was bilateral would suggest a congenital genesis. The cartilage bodies showed very slow growth. However, in the right knee they have begun to separate from the capsule. There seems to be no explanation for this or the fact that it did not happen also in the left knee.

CASE 11. H. E., aged 36 years, No. 2237–28. The patient had been conservatively treated in a hospital for a foreign

Fig. 22.

Fig. 23.

Figs. 22 and 23. Right knee. Small round shadows in the joint cleft and in the back of the capsule. Calcified chondromata in the metaphysis of the humerus.

body in the left knee. The knee at that time had been greatly swollen and could not be fully extended. The patient believed that the lesion was caused by the fact that he had to do most of his work in a kneeling position. In 1914 he suffered a new knee joint effusion. For the past 2 years he has had intermittent pain in the knee and sometimes the onset was so sudden that he could scarcely bend the knee. The patient had noticed on the posterior middle portion of the knee an odd body which protruded but which he could press back himself; when this body had been replaced the pain disappeared. One day early in September, 1928, the patient suddenly arose from the kneeling position whereupon he felt such a sharp pain that he could not extend the knee and could not stand on his left leg. He massaged the knee with a rotating movement and thus succeeded in relieving the condition. These attacks have since that time been repeated as often as 20 to 30 times a day. The patient could always feel an odd body, sometimes at the side, sometimes in the middle of the patella, and sometimes under the quadriceps tendon. The patient had always been successful in eliminating the pain by the movement described. The left upper thigh showed 2 centimeters atrophy as compared with the right. Complete extension was possible, but flexion was moderately restricted. The X-ray photograph showed a round, calcified body in the knee joint cleft (Figs. 26 and 27) and another one in the rear portion of the joint capsule near the head of the fibula. It was supposed that two free bodies were present in the left knee, and operation was advised and carried out in October, 1928. The left knee was opened through a Payr incision, which made possible a close survey of the whole joint. In the upper recess an odd body the size of a cherry was found and removed. A second free body, however, could not be found in spite of the fact that a very close scrutiny of the joint was possible and that a diagnosis of such a body had been made. The second free body was evidently in the joint capsule. The control photographs of 1929 showed that the free body which could

Fig. 24.

Fig. 24.

Fig. 25.

Figs. 24 and 25. Anteroposterior view of both knees and lateral view of right knee. A great number of round not greatly calcified bodies are seen principally on the front of the joints.

not be found at operation still lay in exactly the same position as it did in 1928. The roentgenograms also showed three other round bodies, slightly calcified in nearly the same position as the free body removed at operation. The follow-up examination showed no effusion in the left knee joint, but severe friction with each movement.

The case is interesting for two reasons; (1) The extreme squeezed in appearance caused by a loose cartilage body which made necessary arthrotomy and the removal of the loose body; (2) the follow-up after a year showed a recurrence with an even greater number of capsule chondromata than were present before operation.

Fig. 26.

Fig. 27.

Figs. 26 and 27. Left knee. In the joint cleft is an ovoid, compact cartilage shadow, also a smaller, round body, quite near the head of the fibula.

Case 12. R. G., aged 37 years (patient at the Institute for Physical Therapy). Seven years ago patient was treated for syphilis and was cured. For the past 7 years he has been constantly treated for sciatica of the right side. Acute pains began 3 weeks ago. Day and night he had severe pain, extending from the buttock to below the calf, especially noticeable when he moved in bed. The pain was so severe he had to limp. Reflexes were absent. Adduction and flexion of the right hip were restricted and painful. There was an area of hypæsthesia and a sense of pressure over the adductors. The X-ray picture (Fig. 28) showed foci of calcification about the size of hazelnuts arranged around the capsule. The joint contours were sharp but there were no signs of a secondary arthritis deformans. The bones showed the atrophy of disuse.

Even if this is a case of advanced chondromatosis we have no right to charge to it all the complaints of the patient. The hypæsthetic area over the adductors and the absence of reflexes may be considered as an expression of the syphilis. The fact that the pains were noticeable especially on movement point to an arthritic genesis. The patient could not be located for follow-up.

Case 13. A. E., aged 45 years. Patient had been treated at the Institute for Physical Therapy. In 1908 he lifted a weight too heavy for him. Since then he has always had backache. In 1925 he came to the Institute for Physical Therapy because the pains had become so severe that he could hardly move. It was thought that he had a sciatic neuritis on the right side. In addition there was a paralysis

Fig. 28. Right hip. Calcified chondrom knots up to the size of peas or hazelnuts lie around the joint. The larger show dark borders and clearer centers.

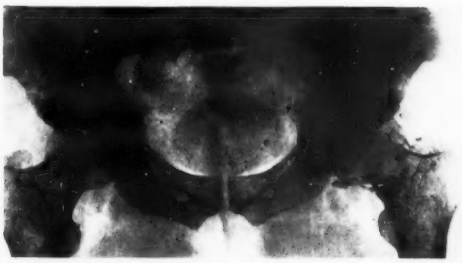

Fig. 29. Right hip. Small calcification foci in the joint capsule and also sharp round calcifications, which must be considered as phleboliths. In the symphysis region there is likewise a foci of calcification probably prostatic calculi.

of the right peroneus, which entirely disappeared after treatment with electrotherapy, but its cause was not learned. Patient was discharged without pain but he re-entered the Institute for Physical Therapy because of a fainting-fit and a spasm of the right calf. The stool at this time showed lead and analysis of the blood revealed punctate basophile erythrocytes. Patient worked in an accumulator factory. He complained of no pain in the right hip. Movements of the right hip were absolutely normal as they were on the left side. Patient walked straight and did not limp.

The X-ray picture taken in 1925 (Fig. 29) showed (as did the follow-up picture) a chondromatosis of the right hip, marked by lentil-sized, clod-like foci of calcification in the capsule. The joint contours were sharp. There were no signs of a secondary arthritis deformans. Besides this there were in the pelvis several circular, sharp foci of calcification which undoubtedly were phleboliths. In the region of the symphysis there was a calcified body of very irregular shape. Whether this was a calculus in the prostate, the roentgenologist could only suppose but could not prove.

This case is very complicated. We have the history of the patient being injured many years ago, of a paralysis of the peroneus which was not explained, and now a proved lead intoxication during the course of which the patient lost consciousness. He had suffered many years with a steady backache and sciatic pains which entirely disappeared. There was furthermore a chondromatosis of the right hip which, however, had not given him the least trouble. We believe that we are right when we say that the chondromatosis had no, or at least no important, rôle in causing these different lesions. X-ray examination of the right hip did not show any variation in the last 4 years, but the hip pain and the radiant pain disappeared entirely. The case shows how cautious one must be in diagnosing areas of calcification near joints.

Case 14. U. F., aged 50 years, No. 380 26. Patient had been at different times in the chirurgical clinic in Zurich and in other hospitals. When he was 25 years old he had an inflammatory process in both hips. In 1916 a great number of cartilage bodies had been removed through a longitudinal incision in the inguinal region. This operation did not relieve the patient. Walking was very painful. The inguinal regions are asymmetrical, and strong, clod-like tumors are palpable in them. The legs are in marked flexion and exterior rotation. Movement at the hips is heavily restricted in all directions. This may easily be understood when the X-ray picture is examined (Fig. 30). On both sides there are calcified chondromata, some of them as large as small apples, partly smooth, partly mulberry shaped, in a very irregular arrangement around the hip joint. There is also a marked arthritis deformans of both hips. The X-ray picture made at different times always showed the same picture.

The case represents a very extreme chondromatosis of both hips. Arthrotomy and elimination of the free bodies brought no relief. The inflammation, if it was an inflammation, of the hip joints reported in the history is no longer evident. It may be supposed that even in this first attack the condition was a beginning chondromatosis.

SUMMARY

This study has disclosed certain facts. It is a fact that something is needed to start pain in the presence of chondromatosis. We have seen badly

Fig. 30. Calcification foci as large as small apples are present around both hip joints, in addition to signs of severe arthritis deformans.

deformed joints, in which a secondary arthritis deformans is already present and probably has been present for some time and yet the history discloses that the patient has been aware of the condition but a few days. On the other hand, we see joints very little deformed and the patients have severe complaints. Some patients show no symptoms at all, and the diagnosis of chondromatosis is given only secondary importance. These variations on one hand depend on local conditions themselves, on the other hand on the physical complaints of the different patients. Among local conditions are the presence or absence of locking of the joint, limitation in movement caused by the restriction of space, and pressure on the capsule and the bones. Perhaps the reports of Seeliger (removal of the hydrogenion values of the synovialfluid toward the acid side in case of arthritis deformans) may cast some light on the subject in this connection. Another factor which must be considered is the matter of compensation with a patient who has accident insurance. If the X-ray picture shows a chondromatosis of a joint which has recently sustained an injury, the insured patient in his desire to receive compensation may be influenced by this fact in stating his complaints.

Further facts may be found in a study of the cases reported. The rate of growth of the individual capsule chondroma and the appearance of new ones vary in the different cases; in every case the growth is very slow. The intensity of the complaints does not coincide with the objective findings.

The principal thing learned is that with conservative treatment and sometimes with no treatment at all, the patients continue to do heavy manual labor. In any event it would seem that patients are more comfortable with a chondromatosis than with an arthritis of the same intensity. It would not be good practice in a patient with arthritis deformans to remove all diseased tissue in a joint or even to reduce the joint itself. On the other hand, if the joint presents a squeezed in appearance operation should be done. Arthrotomy and the removal of the free joint body may be considered but symptomatic therapy. We shall do this operation only when absolutely necessary and when we believe that there is constant danger of recurrence.

CONCLUSIONS

1. Chondromatosis is probably not always caused by the same thing; it may be due to disturbances of development or it may result from chronic irritation of normal cartilage islands of the synovialis. In spite of our knowledge in regard to arthritis deformans, the cause of chondromatosis as a disease entity is still not settled.

2. How great a rôle trauma plays in the etiology must be determined in each case.

3. Chondromatosis may be present and yet produce no symptoms.

4. Chondromata grow very slowly and are never fatal.

5. Treatment must be restricted to healing with the hope that secondary arthritic changes may be prevented. An exception, however, is the case in which operation must be undertaken because function has been interfered with and there is a squeezed in appearance of the joint.

THE DUODENAL NICHE—A CRITERION IN THE HEALING OF DUODENAL ULCER

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THE first to call attention to the value of the roentgen-ray in the study of the changes undergone by an ulcer under medical treatment were Friedenwald and Baetjer. They state that they "can approximately determine the degree of healing of an ulcer which cannot be as certainly determined in any other way."

Öhnell published a series of articles based on results obtained in the medical treatment of gastric ulcer patients from the clinic of Professor Jacobaeus, of Stockholm. He was able to demonstrate the disappearance of a gastric niche by the roentgenological method in most cases, and contended therefore that the assumption that the presence of an ulcer necessarily called for surgical intervention was unfounded. In his experience the average length of time for the disappearance of a niche was 40½ days. The published evidence consists of silhouettes, which show these changes. The actual roentgenograms are not shown.

Hamburger showed actual disappearance of gastric niche formation in the roentgenogram. In one case in which the gastric niche had disappeared, normal peristalsis was not present in that region, indicating the persistence of induration, and that complete restitution to the normal had not occurred. The demonstration of the disappearance of the roentgen-ray evidence of a duodenal deformity is not quite so clear. An important observation made by Hamburger is that although the niche may disappear, a weakness of the wall in that region may persist, and may be the site of recurrence at a later period, with the development of a reactivated ulcer.

Rosenthal published a monograph in 1919 in which excellent roentgenograms of gastric ulcers are shown and the diminution with eventual disappearance of the niche is traced in a large number of cases.

White has noted the disappearance of niche formation in the stomach but has never seen a markedly deformed bulb, representing a duodenal ulcer, return to a perfectly normal rounded contour, in spite of the fact that the patient remains well for a long time and is clinically cured. Such persistent deformity must be considered as due to unyielding scar tissue and adhesions, which, once formed, remain in spite of the actual healing of the ulcer itself.

Buckstein traced the gradual disappearance of a large niche of the lesser curvature of the stomach. Roentgenographic examination was made every 2 weeks, and the retrogressive changes were recorded for a period of 16 weeks.

Shattuck followed the cases that he had been treating, and he found that roentgen observations indicated diminution of deformity corresponding to the improvement of the patients. This was more easily demonstrable in the case of gastric than in duodenal ulcers.

A careful study was made by Diamond. He records the results in 14 cases of gastric ulcer and states that in some patients, under prolonged observation, the niche failed to reappear.

Crohn, Weiskopf, and Aschner reported the results of their observations of 20 cases of gastric ulcer which they followed for periods of from 1 to 3 years. They noted the disappearance of the gastric niche in 10 cases and a failure of diminution in the 8 remaining. The prognosis was influenced by the age of the patient, those in the older individuals showing less tendency to heal because of longer duration of symptoms and sclerosis of the vessels of the stomach and duodenum. The improvement was also affected by the duration of symptoms, healing being more prompt when treatment was instituted shortly after the onset. Several cases showed cyclic remissions, followed by complete recurrence of both symptoms and niche formation. These same authors showed that in 2 cases in which partial gastrectomy was done, a few weeks after medical treatment histological evidence of healing could be demonstrated.

More recently Moutier and Porcher were able to show the complete disappearance of a gastric niche in 4 cases.

Moncrief and Nichols observed 17 cases of gastric ulcer for a period of 2 years. The disappearance and reappearance of the niche in these cases was concomitant with remissions and recurrences of clinical symptoms. No relation was found between the length of the history or the original size of the lesion and the rapidity and permanence of healing.

Assmann, and Chaoul and Stierlin in their classic contributions to gastro-intestinal roentgenology record similar evidence of the value of the roentgen-ray in determining the progress of gastric ulcer healing.

Fig. 1. Case 1. A. H. Duodenal niche present prior to instituting medical treatment.

Thus, though an increasingly large literature has demonstrated the roentgenological disappearance of the niche of a gastric ulcer concomitant with the amelioration of clinical symptoms, there is a paucity of evidence demonstrating similar retrogressive changes in the deformity of a duodenal ulcer.

Two difficulties arise. An irregular deformity of the duodenal bulb other than niche formation, which subsequently shows complete disappearance after medical treatment, may create the suspicion of having originally been due to spasm or possibly to incomplete filling. On the other hand, a persistent irregularity which remains unchanged in spite of complete clinical recovery over a long period of time does not necessarily mean that the ulcer has not healed. Unyielding scar tissue may be responsible for a deformity that remains permanently demonstrable in the radiogram.

A more reliable criterion exists in the study of the changes in the niche of a duodenal ulcer, when this evidence is present. The disappearance of such a niche with clinical cure must be as acceptable as in the case of gastric ulcer.

The duodenal niche as a factor in the diagnosis of duodenal ulcer was first suggested by Barclay.

Haudek (15) called attention to the fact that when the duodenal ulcer is penetrating, a niche similar to that in gastric ulcer can be demonstrated, a constant *Wismuthfleck*.

Bier referred to a case in which a niche with an air bubble was present in the roentgenogram, and at operation an ulcer was found on the anterior superior surface of the duodenum penetrating into the liver.

Kreuzfuchs gave a description of the niche, as a persistent fleck of bismuth, in the beginning of the duodenum, definitely isolated from its surroundings and sharply circumscribed. Although not present in all cases of duodenal ulcer, it is nevertheless pathognomonic of the condition when found.

Rieder stated that a crater-shaped or perforating duodenal ulcer may give roentgen findings similar to those of a callous or penetrating ulcer of the stomach, namely, a niche-like area within the duodenum, with localized tenderness on pressure over this region.

Haudek (16, 17) in 1912 and 1913 emphasized two main points of diagnosis: (1) the presence of duodenal stenosis, which may be a sequel, giving rise to contraction, or, as an associated concomitant phenomenon due to the presence of the ulcer, namely, spasm; (2) the presence of the niche, con-

Fig. 2. Case 1. A. H. Absence of niche one year after medical treatment.

sisting of a bismuth deposit within the crater of the ulcer. This is the most valuable sign because it is the only specific one. Unfortunately, it is not so readily demonstrable as the niche of the gastric ulcer. The other points he refers to are either doubtful or of secondary value. These are localized tenderness, hypermotility, hyperperistalsis, hypertonicity, pyloric fixation, and the persistence of shadows within the duodenum other than the niche.

Cole recognized the roentgenological significance of the direct visualization of the crater of a duodenal ulcer and differentiated between the "bullet hole" and "full face" types.

Akerlund, in his classical monograph, has attempted a detailed analysis of the nature of the duodenal deformity that characterizes ulcer in that region. He divides the factors that go to make up the total irregularity into niche formation, retraction in the region of the niche, the defect on the curvature opposite, the eccentrically placed open pyloric lumen, and the saccular dilatations.

The niche is the most direct evidence of ulcer, and in his series of 109 cases a niche was found to be present in 60 per cent. It is conical in shape and varies in size from the head of a match to half the size of a pea. It may be found in all gradations of the lesion from the penetrating form to the superficial defect without induration and even when epithelialization has occurred. It is generally present on the lesser curvature border: when present on the anterior or posterior wall, it is near that border. Roentgenologically it is seen as springing from the lesser curvature even when the ulcer is actually at some distance from it. Occasionally the niche may be found springing from the base of the bulb, or present directly within the bulb substance, the so-called *en-face* niche. Only rarely is it to be found on the greater curvature side. The demonstration of an actual niche enables the examiner to localize the ulcer with exactness, to determine its relative size, and to employ it as a criterion in determining the effectiveness of medical therapy, in the gradual diminution of its size or complete disappearance.

Fig. 3. Case 2. J. O. Duodenal niche prior to treatment.

Fig. 4. Case 2. J. O. Absence of niche two years after medical treatment.

No account of the significance achieved by the roentgenological demonstration of the niche can be considered complete without reference to the beautifully illustrated article of Berg. He claims to have definitely shown a niche in over 50 per cent of his cases of duodenal ulcer, and considers it the most direct and certain sign upon which a positive diagnosis may be based. With an enviable technique, he has studied the relation of the folds of mucous membrane to the niche. Even in the normal bulb, folds may be demonstrated roentgenologically. In the neighborhood of an ulcer, they may show a radiating convergence.

Fig. 5. Case 3. P. C. Duodenal niche prior to medical treatment.

Fig. 6. Case 3. P. C. Absence of niche one year after medical treatment.

Of great interest are the variations in the size of the niche during the life cycle of an ulcer. During the period of remission of symptoms, either marked diminution or complete disappearance of the niche may be observed. With the recurrence of distress, the niche may reappear.

Carman and Sutherland were able to determine definitely the presence of a niche in only 13 per cent of duodenal ulcers in a large series of roentgen examinations, in spite of careful technique which included fluoroscopic observation through every angle of obliquity both right and left, as well as through close approximation of both anterior and posterior walls by palpation behind the screen. In addition, roentgenograms were made in the angle that showed deformity to best advantage. Of greatest significance is the fact that in spite of this relatively small percentage of cases showing the duodenal niche, of 100 cases that came to operation 98 were correctly diagnosed. Even in the 2 remaining cases, it is possible that the roentgen diagnosis may have been correct, as the duodenum was not opened. In 3 cases of that same series the diagnosis was not verified until the duodenum was opened.

This would signify that while the niche, when present, is as valuable a criterion as in gastric ulcer, in the last analysis the diagnosis depends primarily on the finding of a persistent characteristic deformity which may be entirely independent of any concomitant niche formation. Only when such deformity is absent or doubtful and the niche is found as practically isolated evidence, does it assume its maximum value. Thus, though the demonstration of the niche gives additional certainty to the diagnosis and enables us not only to determine the location of the corresponding ulcer with precision but acts as a guide in the judgment of therapeutic effectiveness, a roentgen diagnosis of duodenal ulcer may be made with remarkable accuracy in its absence.

In our series at Bellevue Hospital with a technique similar to that of Carman, with repeated examinations where the slightest doubt existed, in a definite effort to determine the maximum number of cases in which a niche could be shown, we found it present in 29 per cent of cases of duodenal ulcer.

Three cases are included in which a niche was roentgenologically demonstrable. Following successful treatment with a modified Sippy regimen it was possible to indicate the complete disappearance of the niche for a prolonged period of time, as indicated in the individual case reports, in

spite of frequent and careful fluoroscopic and radiographic examinations.

CASE 1. A. H., aged 26 years, had been complaining of epigastric distress for 18 months. She had had periodic remissions of about one month's duration, only to have the symptoms recur in a more aggravated form. The pain occurred 2 to 3 hours after meals, was localized to the epigastrium, did not radiate, and was relieved by food. She was occasionally awakened at about 2 or 3 in the morning with pain lasting for a few minutes. Once, she was awake all night with pain. She vomited twice, but blood was never found in the vomitus. At one time she noticed blood in the stool. She had lost 20 pounds in weight.

Physical examination was negative except for epigastric tenderness.

Roentgenographic examination revealed the presence of a persistent niche within the duodenal bulb. The stomach itself was normal. No gastric retention of the motor meal was present at 6 hours. A diagnosis of duodenal ulcer was made. The appearance of the niche is clearly seen in the accompanying roentgenogram (Fig. 1). The patient was placed on medical treatment. Her symptoms disappeared. She regained the 20 pounds she had lost. Six months after the first roentgenogram, she was again examined. In spite of the most careful fluoroscopic examination, which was repeated several times, with particular emphasis on manual pressure over the bulb, as well as numerous films, the niche originally noted was no longer demonstrable. Since then frequent examinations have failed to show any recurrence for a period of 1 year.

Such evidence must be considered as objective proof of the healing of duodenal ulcer.

CASE 2. J. O., aged 23 years, had had periodic recurrences of epigastric distress for a period of 3 years. The longest interval of freedom from distress prior to this examination had been 6 months. The distress was epigastric, did not radiate, and occurred a few hours after meals. It was relieved by food. He had never vomited. His bowels were irregular but he had never noticed blood. He had not lost any weight. He gave no history of night pain.

Physical examination revealed tenderness in the right upper quadrant.

Roentgenographic study showed the stomach to be normal. No pathological retention was present. Within the duodenal bulb a niche was demonstrable. This was persistently present during prolonged fluoroscopy and on repetition of the examination. It was also shown in the accompanying roentgenogram (Fig. 3). The evidence of a niche justified the diagnosis of duodenal ulcer. The patient responded readily to medical treatment with complete disappearance of all symptoms, and a moderate gain in weight. Two years after the first roentgen study, during which time he felt fine, he returned for re-examination. At this time, the most careful study, fluoroscopically and with numerous films, failed to reveal the niche which had been present.

When a niche is demonstrable, its disappearance may be noted as a check upon the efficacy of medical treatment. Roentgenologic study thus serves as a valuable means of indicating objectively progressive changes in duodenal ulcer.

CASE 3. P. C., aged 27 years, began to complain of abdominal distress 3 months before her first roentgen-ray examination. The pain was in the epigastrium and occurred when patient felt hungry. On eating, the pain would disappear. The distress was gnawing in character and would recur from 1½ to 2 hours after meals. Frequently she would be awakened at 2:00 in the morning with pain. The ingestion of a glass of milk and some crackers gave her relief in 10 to 15 minutes. She had never vomited or passed tarry stools.

Physical examination disclosed exquisite tenderness to the right of the epigastrium.

Roentgen-ray examination showed a normal stomach. Within the duodenal bulb was a large niche. Tenderness was localized to that region. The niche is clearly shown in the accompanying roentgenogram and represents the crater of an ulcer (Fig. 5). The patient was put to bed and treated conservatively. All her symptoms disappeared. One year after the original examination, she was again roentgenographed. At this time no evidence of the former niche was present. Localized tenderness was absent.

Here again, roentgenological study has shown excellent proof of the healing of a duodenal ulcer concomitant with clinical improvement. It also shows the significance of demonstrating the presence of a niche. The niche enables us to localize the ulcer, determine its relative size, and study its disappearance in the healing process.

SUMMARY AND CONCLUSIONS

1. An historical survey of the significance of the niche in the healing of gastric ulcer is presented.

2. Similar significance from a diagnostic and therapeutic standpoint may be attributed to the niche of duodenal ulcer.

3. Three cases of duodenal ulcer are presented, in which the disappearance of the niche is traced, concomitant with complete amelioration of clinical symptomatology.

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INTRABRONCHIAL DRAINAGE

Its Importance in The Diagnosis and Treatment of Pulmonary Suppurations[1]

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In the study of our cases of lung suppuration and infections which have come to the thoracic surgical clinic at the University of California Hospital, we have attempted to analyze the disease and study its progress by all the usual methods, such as X-ray, bronchoscopy, and diagnostic pneumothorax. We believe that one of the most important factors, which has been frequently overlooked in the study of these cases, is the question of drainage. As has been well said by Yandell Henderson, the lung is the most perfectly drained organ of the body, and its disabilities arise when under any circumstances this drainage becomes deficient.

In our own work we have used the terms "external" and "internal drainage." External drainage is the emptying of a bronchus by cough and expectoration. Internal drainage designates the spilling of pus or secretion from one bronchus into another on the same or the opposite side.

In this paper we propose to show the part that internal drainage plays in the diagnosis and treatment of lung suppurations.

In order to understand internal drainage of the lung and thereby to follow the course of infections, we wish to draw attention to the following anatomical facts:

The carina (Fig. 1) is the vertical partition that marks the bifurcation of the trachea. The carina will permit pus to spill over freely from the right main stem bronchus into the left main bronchus and vice versa (Fig. 3). The right upper lobe bronchus arises from the side wall of the right main stem almost directly across from the carina. About 1½ inches below this upper lobe opening, the right main stem divides into the middle and lower lobe bronchi. The middle and lower lobe bronchi arise together and are separated only by a thin transverse ridge. This free communication permits drainage and spilling of pus from the middle to the lower lobe and vice versa. The middle lobe bronchus runs toward the anterior chest wall, while the lower lobe bronchus passes posteriorly (Figs. 4 and 5).

The left main stem bronchus divides into the left upper and left lower lobe bronchi at a point about 2 inches down from the carina (Figs. 6, 7, 16).

Note the difference in the position of the upper lobe bronchi of the two sides with respect to the carina and lower lobe openings (Fig. 7). The right upper stands out by itself close to the carina, while the left is 2½ inches down from the carina. The right is 2 inches above the lower lobe opening; the left is alongside its lower lobe opening. This difference in the position of the upper lobe bronchi is important from the standpoint of intrabronchial drainage.

A clearer understanding of the intrabronchial anatomy and the unusual facilities for the drainage of pus from one bronchus to another is afforded by bronchoscopic examination and lipiodol studies.

The symptoms and signs in pulmonary suppuration are often more dependent upon the areas to which the pus drains than on the actual site of the primary lung lesion. This drainage in turn depends upon several factors, namely: (1) the patency of the bronchus, (2) the location of the primary lesion, (3) the anatomical relationship of the bronchial openings, (4) the posture of the patient, and (5) the viscosity of the intrabronchial secretion.

If the bronchus leading to the diseased lobe is not patent the pus will be trapped, atelectasis will follow, and there will be no expectoration or drainage to other neighboring bronchi. This plugging of the bronchus may be due either to extrabronchial or intrabronchial conditions. Apart from tumors, the most common causes of intrabronchial plugging in lung suppuration are chunks of thick inspissated pus, intrabronchial granulations, and inflammatory œdema at the bronchial opening.

These latter conditions are frequently transient, and when sufficient pus is welled up behind the plug, "the bronchial opening is forced" and drainage commences. This type of drainage usually is not adequate and bronchial plugging again follows at a later time. There may be several successions of plugging and unplugging at various periods in the course of the disease. When the diseased bronchus is open, however, the areas to which the pus drains will be determined in a large measure by the viscosity of the pus and the posture of the patient.

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[2]J. J. and Nettie Mack Memorial Foundation.

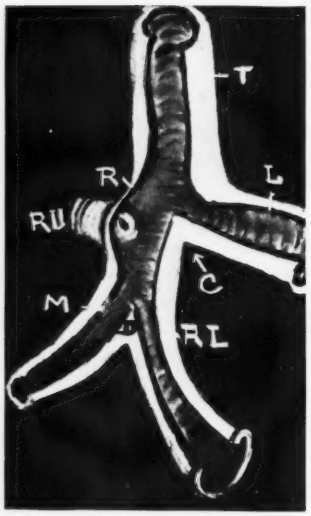

Fig. 1. Diagrammatic sketch of right main bronchi. *T*, Trachea; *C*, carina, *R*, right main stem bronchus; *R. U.*, right upper lobe bronchus; *M*, middle lobe bronchus; *R. L.*, right lower lobe bronchus; *D*, point of division between the bronchial openings to the middle and lower lobes; *L*, left main stem bronchus.

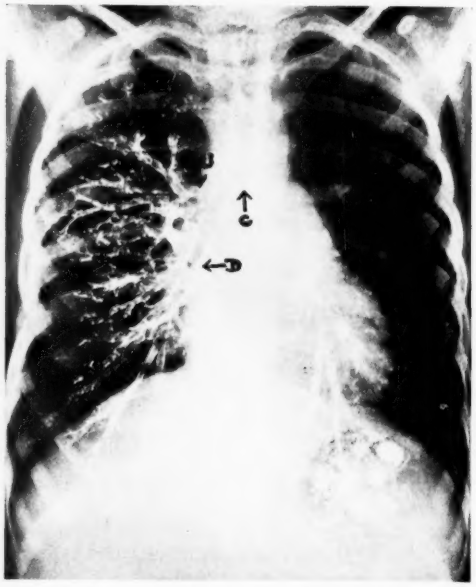

Fig. 2. Lipiodol injection of right tracheobronchial tree. *C*, Carina; *R. U.*, bronchus to upper lobe; *D*, point at which the right main bronchus divides into the middle and lower lobe bronchi. Note the distance between the points *R. U.* and *D*.

MIDDLE LOBE ABSCESS

This relationship of posture and drainage is well illustrated in middle lobe suppurations (Fig. 8).

The middle lobe drains best when the patient lies upon his back, but unfortunately this is an undesirable type of drainage since the pus spills into the right main and lower lobe bronchi. Consequently, the symptoms begin when the patient lies down (Fig. 9). Because of the spilling of pus, the normal air flow is impaired and the vital capacity is suddenly reduced. This initiates severe paroxysms of coughing and expectoration. The sudden severe paroxysms end as soon as the bulk of the pus is expectorated, but a slow continuous drainage from the middle lobe abscess persists. The abscess continues to spill its contents into the neighboring bronchi during sleep. It first fills the dependent portions of the lower lobe and finally accumulates in such quantities that it "wells up" into the main bronchus and thence into the trachea to spill over into the opposite lung. When the vital capacity is sufficiently reduced, the act of breathing becomes a severe effort and extreme dyspnœa, cyanosis, and anxiety ensue.

This picture may last for minutes or hours. Expectoration is hard to start but when once commenced usually gives relief. The attacks leave the patient exhausted.

During the day, however, there is fortunately much less distress than at night. There is little cough and little expectoration since middle lobe drainage and spilling is poor in the erect position (Fig. 8). If the pus accumulates in sufficient quantities to overflow from the middle to lower lobes, there is a sudden attack of dyspnœa and productive coughing, but this is at infrequent intervals. It is not so severe and does not last as long as the morning or "waking" seizures. The number of attacks during the day is dependent upon the size of the abscess, the rate of pus formation, and the adequacy of drainage. Comparative comfort can be maintained if the patient lies on his back at frequent intervals to drain the abscess posteriorly. He may then evacuate the pus from the bronchi by leaning over the side of the bed, in the usual position of postural drainage.

This clinical picture of middle lobe abscess explains the influence of posture and the effects of undesirable "internal drainage." Thus a patient can be observed during the day when he is up and about and temporarily free of symptoms, but

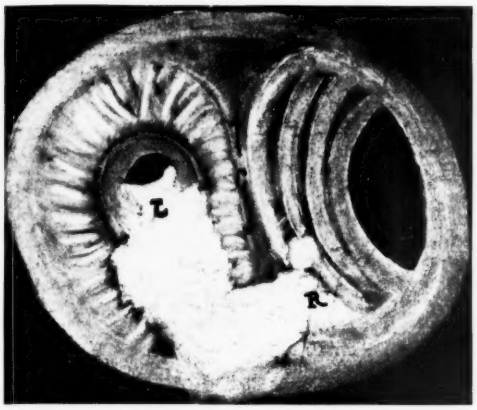

Fig. 3. Bronchoscopic picture showing pus freely spilling from a diseased left main bronchus, *L*, across the carina, *C*, into the right main bronchus, *R*. This is the undesirable internal drainage.

Fig. 4. Iodized oil injection of the right lung (lateral view). *A*, Anterior chest wall; *P*, posterior chest wall; *R. U.*, upper lobe bronchial branches; *M*, middle lobe bronchus; *L*, lower lobe bronchus. The middle lobe bronchus, *M*, and the lower lobe bronchus, *L*, come off the main trunk at the same point, *D*. The upper lobe bronchus, *R. U.*, comes off by itself higher up.

upon lying down the spilling starts and symptoms come on with startling rapidity.

Middle lobe abscesses may give practically no abnormal signs over the middle lobe area if the patient has been lying flat on his back for some time preceding the examination. In such a case the pus will have drained into the lower lobe and, on a basis of physical findings alone, at one examination, lower lobe disease will be diagnosed while middle lobe suppuration will be entirely unsuspected. We have had 3 such cases.

RIGHT UPPER LOBE ABSCESSES

If drainage is sufficiently adequate to keep up a slow steady "spilling" from an upper lobe lesion, the pus will collect in the dependent areas (Fig. 10). One may then encounter evidence suggestive of extensive middle and lower lobe disease with very few signs that point to the real source of the trouble in the upper lobe (Fig. 11). In cases of this type there is a mild expectoration but vital capacity is so gradually diminished that no severe coughing paroxysms occur. Following sudden changes in posture and the rapid spilling of pus to obstruct new areas, the cough and expectoration become extreme (Fig. 12).

The coughing paroxysms in these patients may result from the reduced vital capacity and is not entirely dependent on the amount of pus within the tracheobronchial tree.

Lipiodol injections teach us considerable concerning the factors which govern these paroxysms.

These lipiodol observations are not made on cocainized patients wherein anæsthesia and abolition of the cough reflex would account for the spilling from lobe to lobe. On the contrary, the oil can easily be introduced into the lung by the passive method without any type of anæsthesia and still not initiate cough. But if the patient is suddenly tipped upside down after the oil has collected in the lower lobes, the lipiodol will flow up into the trachea *en masse* and obstruct the entrance and exit of air so that a paroxysm of coughing follows. These masses of oil can be seen under a fluoroscope or on the X-ray plate (Fig. 13). The cough is not due to irritation of tracheal mucous membrane since it will occur if the oil wells up in the trachea even in a patient who has been thoroughly cocainized. This relationship of cough and the sudden interference with the normal air flow is particularly striking during bronchoscopic examinations. Bronchoscopically, a cocainized main bronchus can be touched at numerous points at random with a bronchoscopic sponge without exciting cough, but if pus is seen to flow from the depths of the lung so as to occlude this particular bronchus, coughing commences and continues until the pus is dislodged or removed mechanically through the bronchoscope.

Figure 13 shows a mass of lipiodol in the trachea which caused the patient to cough. We have seen this both in cocainized and uncocainized patients. This is, therefore, not a matter of irritability of the tracheal and bronchial mucous membrane but a question of a sudden obstruction of the normal

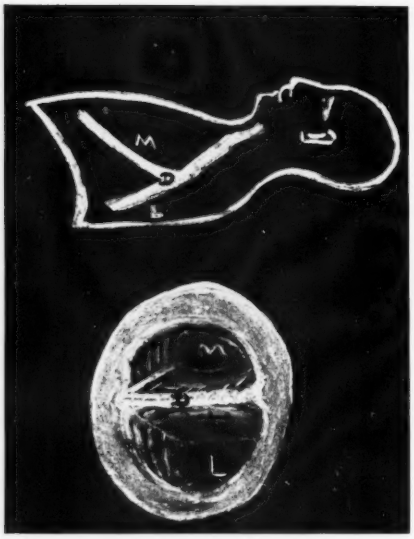

Fig. 5. Upper sketch shows the general direction of the middle and lower lobe bronchi. The middle lobe bronchus, *M*, runs toward the anterior chest wall, while the lower lobe bronchus, *L*, passes posteriorly. Lower sketch gives a bronchoscopic view of middle and lower lobe openings. *D*, Point at which the middle and lower lobe bronchi join to form the main bronchial stem.

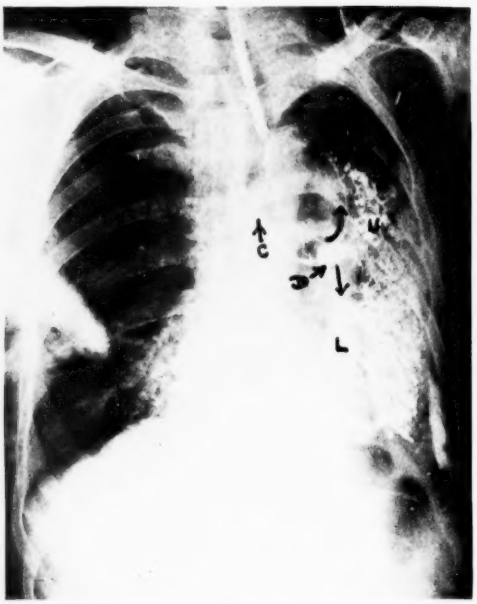

Fig. 6. Lipiodol injection of left lung showing the main anatomical divisions of the bronchi. *C*, Carina; *D*, point of division between the bronchus to the upper lobe, *U*, and lower lobe, *L*. The two unlettered arrows indicate the course of these two main bronchi.

air channels with a resultant reduction in the vital capacity. Twenty cubic centimeters of lipiodol slowly introduced causes no cough, but 5 cubic centimeters rapidly introduced, is sufficient to initiate a paroxysm.

Small amounts of pus near the opening of the right main bronchus can shut off the air to all three lobes of the right lung and reduce the vital capacity to a marked degree, while rather large collections of pus in a single lobe at the base can be well tolerated, as air can then freely enter the remaining portions of this lung.

The "head down" position will evacuate pus that has spilled to the middle and lower lobes during the day. This posture, however, usually leaves the upper lobe abscess undrained except in abscesses situated at the base of the upper lobe (Fig. 14). If the patient lies on his right (affected) side during the night, the abscess cavity completely fills with pus and slowly overflows into the dependent bronchi. When the patient awakens, one or more of the adjacent bronchi will be so completely filled as to prevent the entrance of air. At this time dyspnœa and wheezing will be marked, the patient will be completely choked up, and the coughing and expectoration will be violent and persistent. These symptoms will continue until the pus can be dislodged from those areas that most seriously embarrass breathing.

RIGHT LOWER LOBE ABSCESSES

It is not easy to diagnose positively a right lower lobe lesion on the basis of abnormal lower lobe signs. We have seen several patients who, on careful examination, showed evidence of disease at the base and still, after lipiodol injection, bronchoscopy, and operation, were proved to have primary upper lobe lesions even though no abnormal physical findings were elicited over the upper lobe (Fig. 11). Examination with the patient in different positions at various times during the day and night would have prevented this error since the location of physical findings in these cases is determined by the spilling due to posture.

The deductions to be drawn from physical findings are to be based upon several examinations at various times of the day or on separate days after the patient has assumed various postures for several hours preceding each examination.

Fig. 7. Anatomical specimen showing the anatomy of the tracheobronchial tree. P^1, Left lung; P^2, right lung; *C*, carina; *R. U.*, right upper bronchial opening; *L. U.*, left upper lobe bronchial opening; *D*, division between the right middle, *M*, and lower lobe bronchi, *R. L.*; *T*, trachea; *R*, right main bronchus; *L*, left main bronchus; *L. L.*, left lower lobe bronchial opening. The distance between the right upper and lower bronchial openings is not so great here as is usually the case.

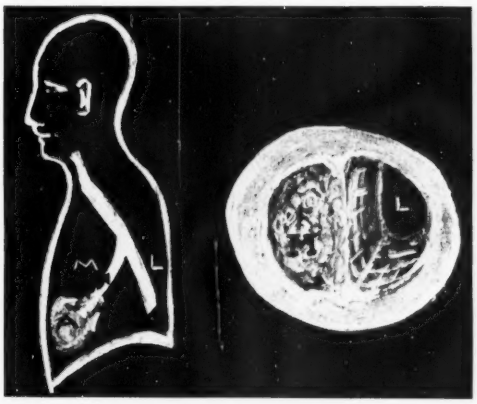

Fig. 8. A sketch and bronchoscopic view of a middle lobe abscess with patient in the erect position. *M*, Pus filled middle lobe bronchus; *L*, normal lower lobe bronchus; *A*, abscess of the middle lobe is close to the anterior chest wall. In this erect position, pus from the middle lobe abscess must "flow up hill" in order to drain. Therefore drainage and spilling are slight.

LEFT UPPER LOBE ABSCESSES

Abscesses at this location follow the same general plan of spilling that has already been noted. Because of the nearness of the main bronchial openings on the left, pus spills from left upper lobe abscesses into the left lower lobe to give abnormal physical signs over this lower region (Fig. 7). Pus can then drain across from the left lower lobe into the opposite lung (Fig. 15).

We have recently seen a proved case of left upper lobe abscess in which there were just as many abnormal signs over the lower lobe as over the diseased upper lobe. Two other cases of this type have been studied. One was a boy of 16 years. He was seen because of a chest complication 2 days after an appendectomy. When the patient was examined lying flat on his back, all the abnormal physical findings were limited to the upper lobe. The lower lobe findings were normal. As soon as the patient was placed in an extreme Fowler position, the abnormal signs disappeared from the top; but an impaired percussion note, along with râles and diminished breath sounds, was noted over the lower lobe which had previously been perfectly clear. This migration of signs occurred almost instantaneously with the change of position. The third case of migrating signs occurred in an elderly woman 2 days after a hip fracture. The initial examination in the flat position showed all the abnormal signs over the left upper lobe (front and back), while the lower

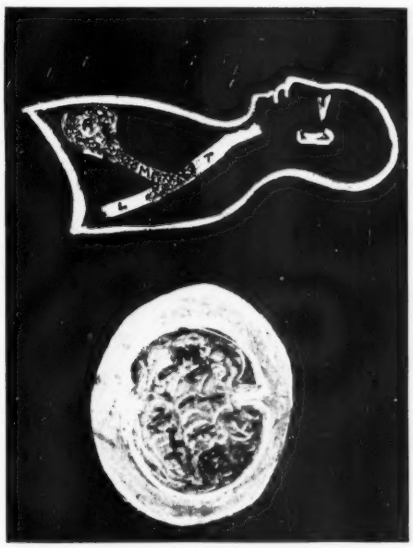

Fig. 9. Sketch to illustrate the influence of posture on the drainage of middle lobe abscesses. Note pus spilling from the middle lobe bronchus, *M*, into the lower lobe bronchus, *L*. *A*, Abscess of middle lobe. Lower sketch shows bronchoscopic appearance of the pus filled middle, *M*, and lower, *L*, lobe bronchial openings with the patient lying upon his back. This interferes with the entrance of air to these two lobes.

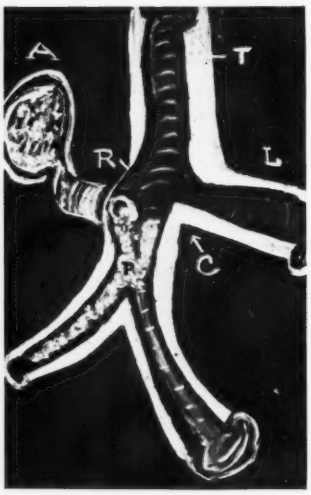

Fig. 10. Diagram showing sites to which pus spills from an upper lobe abscess if the upper lobe bronchus is open and the patient is erect. *A*, Right upper lobe abscess; *R*, right main stem bronchus; *C*, carina; *L*, left main stem bronchus; *P*, pus collecting in the middle and lower lobe bronchi. One bronchus is fairly well filled up with pus and will give physical signs of a drowned lobe or possibly atelectasis, while the other bronchus has only its mouth occluded. The signs over this latter lobe will suggest an obstructive emphysema. If the bronchial opening is only partially occluded with pus, there will be an audible expiratory and inspiratory wheeze.

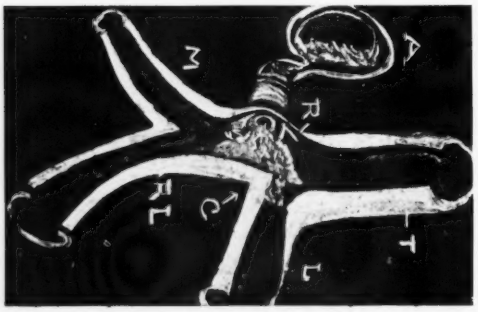

Fig. 12. Diagram of pus spilling from a right upper lobe abscess when patient lies on his left side so as not only to obstruct the right main bronchus, *R*, but also to occlude the left main bronchus, *L*. Pus has spilled across the carina, *C*. The entire lung is not filled with pus but the two main bronchi are occluded and no air can enter either lung. Vital capacity is suddenly reduced and cough ensues. All degrees and varieties of this spilling can be encountered. Physical signs of partial obstructive emphysema with inspiratory and expiratory wheezes are noted over both lung fields. Patients with right upper lobe abscesses cannot lie on their left sides because of this constant drainage and spilling into the left bronchus.

Fig. 11. Iodized oil injection of right lung. The arrow indicates the dilated bronchi at the base of the right upper lobe. A previous roentgenogram without lipiodol showed no evidence of right upper lobe disease. All the abnormal physical findings were over the middle and lower lobes. The location of these abnormal physical signs can be explained on a basis of "internal drainage" and spilling of pus from above. Operation confirmed the lipiodol diagnosis of upper lobe bronchiectasis and showed a normal middle and lower lobe.

lobe was normal. After assuming the Fowler position and instituting deep breathing exercises, the left upper lobe cleared up instantaneously but the râles appeared over the lower lobe. In these 3 cases, posture and drainage have been the influencing factors in determining the location of abnormal physical signs.

LEFT LOWER LOBE SUPPURATION

Left lower lobe abscesses spill their contents into the left upper lobe when the patient lies on his affected side, but the pus spills across the carina into the right upper lobe when patient lies on the right side. This tendency to secondary right upper lobe involvement is due to the high anatomical position of the right upper lobe bronchus (Figs. 1 and 7).

Lipiodol injections in unanæsthetized patients illustrate very well the susceptibility of the right upper lobe to accumulate pus from the left lower lobe (Fig. 15).

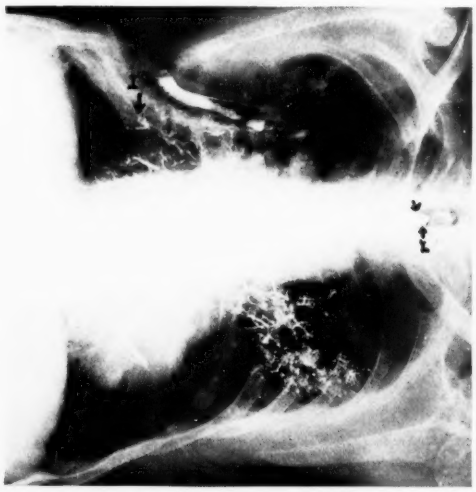

Fig. 13. Lipiodol introduced into unanæsthetized bronchial fistula of right lower lobe with patient lying on left side. Note the spilling of the lipiodol over from the right lower into the left upper lobe. *I*, Point of injection through bronchial fistula; *L*, chunk of lipiodol in the trachea.

Fig. 14. Lung abscess of right upper lobe that best evacuated itself in the "head down" position. Arrow indicates the "mouth" of the bronchus leading to the abscess. Right upper lobe abscesses usually drain best when patient is erect rather than down.

On bronchoscopy, we have seen 2 patients with chronic left lower lobe suppuration in whom there was a collection of pus along with marked redness and œdema at the right upper lobe bronchial opening. These right upper lobe bronchoscopic observations might so mislead one as to make a diagnosis of primary right upper lobe disease. In these cases, however, the right upper lobe was normal on lipiodol examination and the abnormal bronchoscopic changes in the right bronchus must therefore be interpreted on a basis of "spilling" due to the intrabronchial drainage. The bronchoscopic examinations and lipiodol injections have been decidedly helpful in interpreting and evaluating the physical signs.

The physical findings in lung suppurations are similar to those noted in patients after lipiodol injection and in each instance alter in position after changes in the posture of the patient. These findings must, therefore, be on a basis of fluid or secretion (pus) in the tracheobronchial tree. Lipiodol studies and the accompanying roentgenograms give us a fair conception as to the mechanism of intrabronchial drainage. The areas to which lipiodol can be spilled on changes of posture are identical with those at which abnormal physical findings are elicited in lung suppurations following similar postural changes (Figs. 13 and 15 to 19). Lipiodol introduced into the left upper

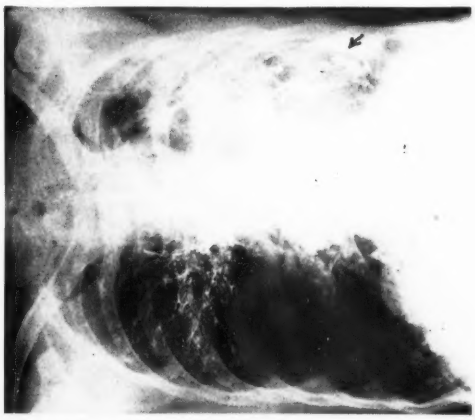

Fig. 15. Lipiodol introduced into a bronchial fistula of the left lower lobe without the use of cocaine or any type of anæsthesia. Patient is lying on his right side. Note the flow of lipiodol into the opposite lung plainly to fill the right upper lobe. There were no râles over the right upper before lipiodol injection; coarse moist râles appeared after the lipiodol "spillover." Pus will likewise spill from the left lower lobe into the right upper with the resulting production of abnormal physical signs. This illustrates the influence of posture on intrabronchial drainage.

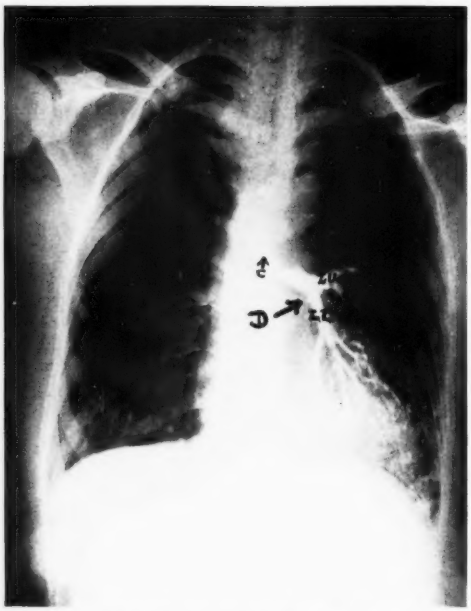

Fig. 16. Lipiodol introduced by "passive method" (through the mouth) without any type of anæsthesia, into the left lung with the patient in erect position. Note the absence of lipiodol in right lung. *C*, Carina; *L. U.*, left upper lobe bronchus; *L. L.*, left lower lobe bronchus; *D*, division between upper and lower lobe bronchial openings.

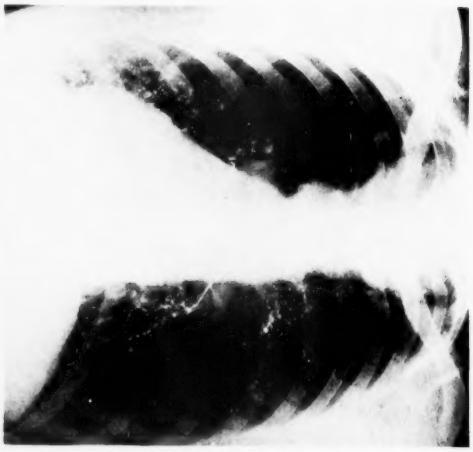

Fig. 17. Same patient as in Figure 16. Roentgenogram taken after patient lay on the right side for 20 minutes. The lipiodol has "spilled" over from the left lung into the right upper and lower lobes. Pus will spill over in similar fashion. This illustrates the undesirable "internal drainage."

lobe will flow to the left lower lobe when the patient assumes the erect position, and similarly the migration of abnormal physical findings from the left upper to the left lower lobe has already been demonstrated in 3 cases of intrabronchial secretion without lipiodol.

With the X-ray we can follow the "lipiodol spilling" from one portion of the lung to another with changes in the posture of the patient; but this cannot be so easily done with pus alone, which does not cast a shadow comparable to lipiodol. However, since many lung suppurations and lipiodol cases have similar physical findings and identical alterations in the findings on postural changes, it seems fair to conclude that the "spilling" takes place in each instance. Bronchoscopic examinations confirm this conclusion.

PRACTICAL APPLICATION

This undesirable "internal drainage" is not limited to lung suppurations; but is noted also in certain cases of pneumonia, tuberculosis, sinusitis, and carcinoma of the œsophagus. The recognition of this type of drainage is of a decidedly practical value in the management of these cases.

Internists are familiar with the so-called migrating pneumonias. It is noteworthy that these often occur at a right top and then at the left base. Because of the anatomical arrangement, it is easy for pus to spill from a right upper lobe bronchus across the carina into the left lower lobe. Internal drainage seems to account for these new areas of pneumonia in such cases. We are studying these conditions at the present time and believe that many of them are due to "spill over" from the original upper lobe lesion.

Thoracic surgeons are cognizant of the unusually poor gas or ether anæsthesia taken by certain lung abscess patients. This is particularly true in patients who have copious expectoration. During the operation the patient is usually lying with the diseased side uppermost, and this permits pus to spill across the carina into the dependent opposite lung so as to obstruct the air flow to the good side. Rapid breathing and cyanosis ensue, and if the pus is not dislodged, death will follow. If the patient is unable to expectorate the accumulated pus because of weakness, pain, narcosis, or lack of support of the chest wall after a first stage lung abscess operation, this "spill over" to the opposite lung will occur. This has been clearly demonstrated in a patient with lung abscess (300 cubic centimeters expectoration in 24 hours) who had a

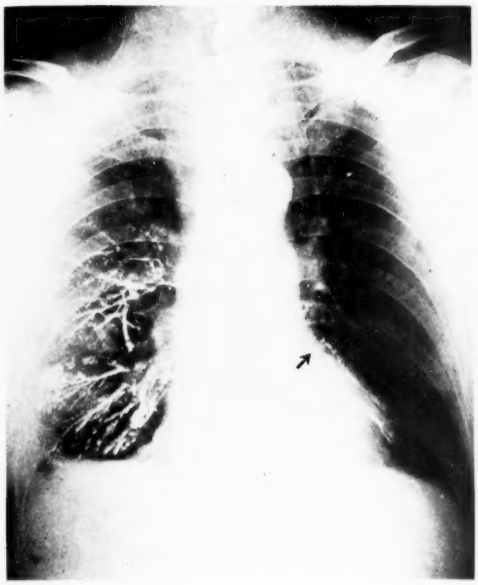

Fig. 18. Lipiodol introduced by the passive method (through the mouth) without any type of anæsthesia into the right lung with patient in erect position. There is a small amount of oil at the left base.

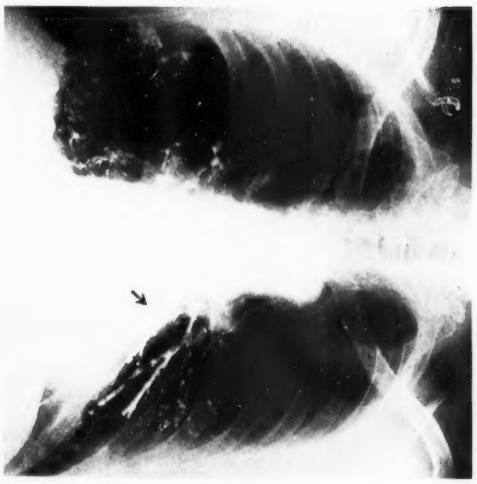

Fig. 19. Same patient as in Figure 18. Roentgenogram taken after patient had been lying on the left side for an hour. The lipiodol has spilled from the right lung into the left side.

first stage lung abscess operation with multiple rib resections and suture of the lung to the chest wall without open drainage of the abscess itself. Morphine and scopolamine anæsthesia had been used. The patient had great difficulty in evacuating the pus postoperatively. He became cyanotic and respirations were rapid. Coarse râles (inspiratory and expiratory rhonchi) appeared over both lungs. He died on the third day. At autopsy, in addition to the pus in the diseased right lung, the left main bronchus and left lower lobe were also filled with pus. Before operation the left lung was clear, and this left sided pus must be explained on a basis of overflow from the right side, especially since no abscess was found on the left side at autopsy.

A patient with a right sided lung abscess, operated upon by cautery pneumonectomy, did beautifully until the third day after operation except for a slight amount of bleeding from the wound which seemed to be controlled by packing. On the fourth day, however, this patient died suddenly and unexpectedly. Autopsy showed almost no blood in the bronchi of the diseased lung but there was a large marble sized clot in the opposite main bronchus with an obstructive emphysema of the entire left lung. Death was due to strangulation, since no air could enter the left lung and the diseased right lung was inadequate to maintain life. Undesirable intrabronchial drainage explains this case of bronchial obstruction. The bleeding from the right lung resulted in a clot which was spilled over or aspirated from the right main bronchus into the left. Bronchoscopy would probably have saved both of these patients.

Before chest operations on lung abscesses, we now examine and treat these patients bronchoscopically to prevent such intrabronchial spilling. Bronchoscopy is repeated at the end of the operation and as often thereafter as necessary.

Pulmonary tuberculosis affords further examples of the practical importance of intrabronchial drainage. Often after a pulmonary hæmorrhage there is an extension of the areas of abnormal physical signs. The blood probably spills across the carina into the opposite lung, since abnormal physical signs appear over both sides and, on a basis of physical findings alone, one is at a loss in deciding on which side to induce an artificial pneumothorax. This is especially true if the patient is seen for the first time during the course of a hæmorrhage.

The problems of intrabronchial spilling are prominent in the operation of thoracoplasty for pulmonary tuberculosis. In the literature, one cannot help noting the difference of opinion which prevails concerning what should be done at the

initial operation. Some prefer removal of the upper ribs, and others favor resection of the lower ribs at the first stage operation. Removal of the upper ribs at the outset occasionally results in a bronchogenic extension of the disease to the uncollapsed lower lobes. This discussion as to choice of operative procedure resolves itself into one of intrabronchial drainage. Bronchoscopy with removal of the intrabronchial pus either immediately before or after the operation would prevent this spilling and extension of the disease.

The close association of paranasal sinus disease and bronchiectasis has been noted frequently and is considered as due to both lymphatic and bronchogenic extension from the sinuses to the lungs. This bronchogenic extension of nasal secretions during sleep has been well shown with lipiodol by Quinn and Meyer. The free communication between the two lungs permits the spilling of the nasal drippings from one lung to the other and may result in bilateral bronchiectasis.

Carcinomata of the œsophagus furnish additional examples for the study of intrabronchial drainage. In these cases, it is not unusual to find evidence of partially or totally occluded minor bronchi at the posterior portions of the right lower lobe and at the anterior lateral portions of the left lower lobe. The pulmonary signs on the right are in back, while those on the left are in front. These signs are due to the œsophageal secretions spilling down over the interarytenoid space into the trachea. The secretions then pass most readily into the dependent areas usually to the posterior part of the right lower lobe. When the patient lies on his left side, however, the anterolateral portion of the left lower lobe becomes the most dependent area. We have noted this bilateral distribution of abnormal lung signs in at least 6 cases of carcinoma of the œsophagus and have interpreted it on a basis of intrabronchial drainage.

In a later paper we will present a series of cases of lung suppuration showing the result of treatment based on intrabronchial drainage. Suffice it to say, however, that the employment of treatment along these lines has increased the percentage of cure and decidedly lessened the number of cases requiring open operation.

SUMMARY

1. We feel that proper evaluation has not been given to the mechanical factors which are present in nearly every case of pulmonary suppuration.

2. Intrabronchial drainage has considerable influence on the production of symptoms, course of the disease, its extension both locally and to the other side, and modifies very materially the physical signs which are found from time to time.

3. The diagnosis and successful treatment of pulmonary suppuration are based on an understanding of intrabronchial drainage.

4. Lipiodol and bronchoscopic examinations permit a study of the factors which control this type of drainage.

5. The symptoms and signs in pulmonary suppuration are often more dependent upon the areas to which the pus drains than on the actual site of the primary lung lesion.

6. The part bronchoscopy plays in the diagnosis and treatment of these conditions cannot be overemphasized.

7. We do not undervalue the many other factors which control and modify lung suppurations, such as the type of infection, its virulence, and the method of extension along the lymphatics and the blood stream.

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PRIMARY CARCINOMA OF THE FALLOPIAN TUBES

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MOST new-growths of the fallopian tubes—papillomata, sarcomata, lymphangiomata, fibromata, and carcinomata—are rather infrequent and the greatest majority are recognized only after the abdomen is opened. Their symptomatology and findings are so similar to that of tubal inflammatory processes that differentiation before operation is seldom made. It is the intention of the authors of this paper to discuss the subject of carcinoma of the fallopian tube and to emphasize the symptomatology and findings before and at the time of operation in the hope that more cases will be recognized and properly cared for surgically.

The first authentic case of primary carcinoma of the fallopian tube was reported by Orthmann in 1888. Doran, in 1910, completed an analysis of an additional 32 cases. In 1926 Wechsler, in an excellent review of the literature, summarized a collection of 192 cases and added 4 of his own. With the cases reported in the past few years it is safe to conclude that there are well over 200 authentic cases on record.

Norris found one case of primary carcinoma of the tube as against 94 uterine cancers in 2,020 gynecological specimens. Vest found only 4 cases of tubal carcinoma in 19,000 patients treated in the gynecological clinic at Johns Hopkins Hospital. In a recent article Callahan, et al., emphasized the rarity of this condition with the following statistics: primary carcinoma of the fallopian tube was found in 0.02 per cent of all gynecological admissions at Johns Hopkins Hospital, in 0.07 per cent of all gynecological admissions at Lenox Hill Hospital, and in 0.31 per cent of all gynecological laparotomies at the Leipzig clinic.

ETIOLOGY

The majority of reported cases of primary carcinoma of the fallopian tube have occurred at about the time of the menopause. Sixty-six per cent of the cases in Wechsler's series occurred between the ages of 40 and 55. The youngest patient (Bower and Clark) reported was 25 years of age, and the oldest (Mantel) was 73.

The etiology of this condition has been a subject of much interest and some disagreement. The theory that primary carcinoma of the tube arises upon the basis of a chronic, probably purulent, inflammation has been generally accredited to Sanger and Barth. Doran and others have expressed the opinion that tubal carcinoma represents a malignant change which has taken place in a benign papilloma. Bower and Clark and Norris have stated that in a large proportion of these women there was a history of long standing pelvic inflammation. Contrary to these statements Wechsler found that in only 8 per cent of 196 cases was there any mention of previous pelvic imflammation. He added that gonorrhœal infection was rarely admitted. Vest, in an attempt to ascertain whether or not the patients had suffered from tubal inflammation, very carefully studied case reports, which included microscopic descriptions, and concluded that inflammation of the tube played a minor rôle, if any, in the development of carcinoma of the tube.

As further evidence of pelvic inflammation, attention has been directed to the relative frequency of sterility, miscarriage, and abortion in these women. Vest, Wechsler, and others attempted to analyze the statistics appertaining to this phase of the subject, but they were unable to draw any satisfactory conclusions.

Callahan recently reported a case of primary carcinoma of the tube associated with tuberculosis of the same organ. He found only 6 other authentic cases in the literature and concluded that the association was coincidental.

Biar, in his thesis, discussed the possibility of cancer being produced by the excitation of wolffian remnants located in the tube and subjacent structures. Gittleson and others have described cases of tubal cancer which appear to have been secondary to parovarian new-growths. The embryonic rest excitation theory has been employed by a number of authors in an attempt to explain the presence of pelvic new-growths of unknown etiology. This theory, however, cannot be applied to carcinoma of the tube as the microscopic pictures, especially in the early cases, clearly indicate a primary disturbance in the epithelium of the tubal lumen.

The frequent co-existence of tumors of the ovary and the uterus with carcinoma of the tube has been considered from the standpoint of etiology. Kittler's patient, in addition to cancer of the right tube, had a dermoid cyst of the right ovary, a simple cyst of the left ovary, and a myomatous uterus. Other similar cases have

Fig. 1. Distal half of tube opened revealing hæmorrhagic papillomatous growth occupying the entire lumen. The proximal half of the tube was patent.

been reported. In his series of 196 cases, Wechsler found the following: cancer in a tubo-ovarian cyst in 10 per cent; a large ovarian cyst separate from the tubal cancer in 8 per cent; a hydrosalpinx of the opposite tube in 10 per cent; and a myomatous uterus in 15 per cent of the cases. There is no evidence, however, to show that the condition of adjacent structures has had any influence on the tubal epithelium which might have led to the development of a new-growth.

It is obvious that the foregoing does not permit the drawing of conclusions concerning the etiology of this condition. That evidences of chronic salpingitis are frequently found in association with this disease can not be denied, but it no more explains the etiology than does a chronic infection of the cervix uteri explain the presence of cancer in that organ. The fact that malignant tumors furnish a very fertile media for the growth and multiplication of pyogenic organisms may serve to explain the presence of inflammation in many cases. It is significant that in a number of cases, the present case included, the only evidences of inflammation are confined to that portion of the tube which embraces the tumor.

PATHOLOGY

Macroscopic. When viewed at operation, tubal cancer usually has the microscopic appearance of a chronic inflammatory condition of one or both tubes, the disease being bilateral in about one-third of the cases reported. The middle and outer thirds of the tube are generally enlarged to form an oval, retort, or sausage shaped mass resembling a hydrosalpinx or a pyosalpinx. The abdominal ostium of the tube is usually closed and not infrequently adherent to the ovary to form a tubo-ovarian mass. The surface is rough or smooth depending on the presence or absence of adhesions or the penetration of the serosa by the growth. The tumor may be cystic or solid with a consistency which varies with the character of its contents. When the tube is opened the lumen is found to be more or less occupied by a cauliflower-like, papillary, or granular friable mass. The wall is usually thickened but may be very thin when associated with a hydrosalpinx or a tubo-ovarian cyst. The fluid content varies in quantity and quality and may be clear, watery, hæmorrhagic, or purulent. Andrews describes a case in which the tumor (tubo-ovarian) contained two quarts of smoky, blood stained fluid.

Metastasis to the retroperitoneal lymph nodes is common and has been noted in some of the very early cases. Peritoneal metastasis is generally limited to those cases in which cancer cells escape through the abdominal ostium or by penetration of the serosa. Direct extension to contiguous structures, expecially ovary and uterus, is frequently noted. Spencer describes a case in which there was a small metastatic nodule in the vagina, this structure and the tube being the only organs involved.

Microscopic. The histology of this disease is well established and pathologists generally agree on two main types—(1) papillary and (2) papillary-alveolar—though various minor modifications of these two patterns are occasionally added. Some authors are of the opinion that the papillary type is original and that the alveolar formation is gradually developed by the fusion of adjacent papillary folds. A few cases (Friedenheim), purely alveolar in character, have been described, but the rarity of this type together with the difference of opinion among authors concerning its origin does not favor its inclusion as a special type.

Except in very early cases it is difficult to reproduce a differential picture of the two main

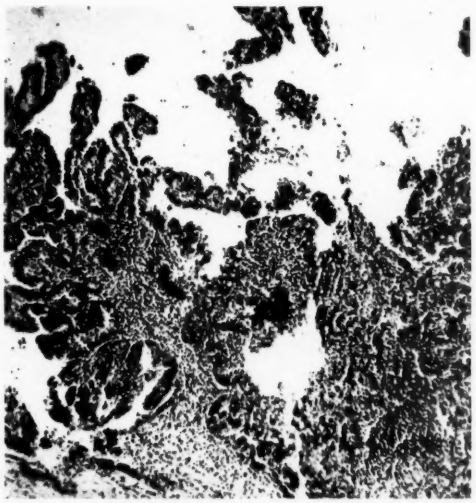

Fig. 2. Section through the tumor wall showing the papillary pattern of the proliferating mucous folds. The round cell infiltration present is rather more marked than in most sections.

types. With epithelial proliferation the papillæ of adjacent mucosal folds intermingle and cohere to form pseudo-alveoli at a very early stage. Many of the affected mucosal folds show marked hypertrophy and arborescent branching consisting of a delicate fibrous tissue core covered with single and multiple layers of cuboidal or low columnar epithelial cells which are irregular in size, shape, and staining reaction. In advanced cases, the mucosal folds and alveolar spaces become distorted as a result of fluid pressure or cellular proliferation or both. The lumen in the solid or semi-solid tumors is occupied by medullary masses, separated by sparse fibrous tissue septa, in which areas of hæmorrhage and necrosis are often observed. Invasion of the muscularis as well as the tubal lumen is occasionally noted, though penetration of the serosa is uncommon even in advanced cases.

Round cell infiltration and capillary dilatation in the mucosa and submucosal layers are noted in a majority of cases. These findings, in some instances, are confined to the affected portion of the tube and are undoubtedly secondary to the development of the tumor and can in no wise be considered evidences of previous inflammation. In our case the opposite tube and the inner third of the affected tube are free from evidences of inflammation.

The subjacent ovary is frequently involved and almost invariably so when the mass is tubo-ovarian in character. The ovarian metastases are generally in the form of medullary masses which occupy cystic spaces or invade the stroma.

SYMPTOMS

There are no characteristic symptoms which in a majority of cases should lead one to suspect a primary tubal malignancy. Vaginal discharge, abdominal pain, and menstrual irregularities are the subjects of common complaint in these women. The discharge, usually watery and clear or straw colored at first, is the nearest approach to a characteristic symptom. It is interesting to note that in the case reported the onset was marked by a bloodtinged watery discharge which, after 5 weeks, became colorless and stainless. The character of the discharge may be modified by infection, necrosis, or hæmorrhage at the site of the growth, or by co-existing pathology in the uterus and vagina. The discharge may be continuous or intermittent, scanty or profuse, depending probably on the patency of the uterine end of the tube. The discharge in our case was continuous and exceedingly profuse. The intermittent type (also, said to be characteristic of "hydrops tubæ profluens" (7), often occurs in periodic gushes and is proceeded and sometimes

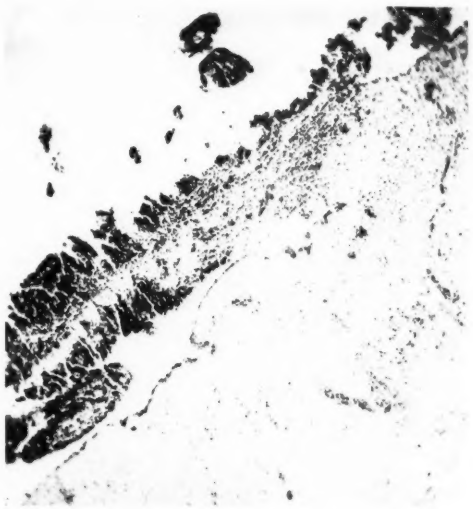

Fig. 3. Section through the tumor wall showing marked hyperplasia of the epithelium covering a mucosal fold. Compare with Figure 2, with regard to round cell infiltration.

accompanied by paroxysmal cramp-like pains which gradually subside as the fluid escapes.

In addition to the pain previously mentioned pain is generally present in the lower abdomen, usually on the side of the lesion. The pain varies in character and intensity from an occasional dull ache to a frequent sharp shooting exacerbation, which very frequently radiates to the back and produces, in some cases, severe sacral backache.

Metrorrhagia is probably the most frequent complaint, but there is nothing typical to distinguish it from bleeding due to uterine pathology. Menorrhagia, dysmenorrhœa, and irregular intervals of amenorrhœa are not uncommon. In evaluating these menstrual alterations the average age of these women must be considered.

Urinary disturbances, gastro-intestinal symptoms, general weakness, and loss of weight are not uncommon during the later stages of the disease.

PHYSICAL SIGNS

There are no physical signs sufficiently characteristic to differentiate tubal malignancy from adnexal disease in general. A tense cystic or solid mass, oval or sausage shaped, varying in size from a few to 15 or 20 centimeters in diameter, is generally felt on one or both sides of the uterus or in the cul-de-sac of Douglas. On the other hand, slight induration in one fornix may be the only finding. When adherent to the ovary the size of the mass will then depend on the condition of that organ. Biar states that the tube is free nearly as often as it is adherent. Ascites is noted in a small number of cases and does not appear, as a rule, until rather late in the disease. Inguinal and supraclavicular lymph nodes are occasionally involved. Anæmia and undernourishment are not uncommon, but extreme cachexia is rare.

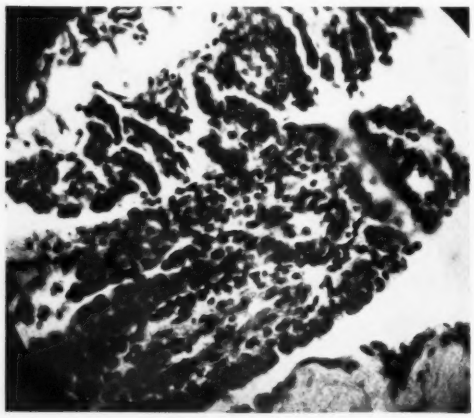

Fig. 4. High power view of lower left corner of Figure 3, showing the polymorphous character of the hyperplastic cells.

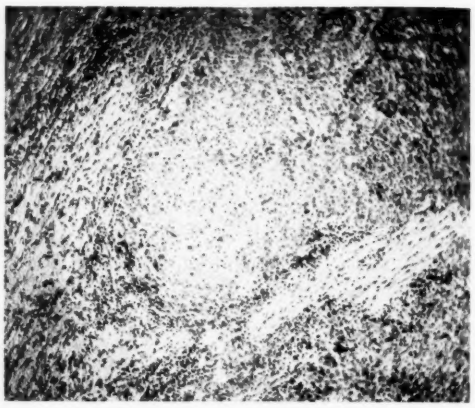

Fig. 5. Section through the tumor wall showing invasion of the muscularis by the tumor cells.

DIAGNOSIS

The insidious onset with signs and symptoms commonly present in various other pelvic conditions makes the diagnosis exceedingly difficult. In only a few instances is cancer of the tube mentioned in pre-operative diagnoses. This was based upon the microscopic appearance of tissue obtained by a diagnostic puncture of the cul-de-sac. Chronic pelvic inflammatory disease, tubo-ovarian abscess or cyst, cancer of the ovary or the uterus, and myoma uteri are the conditions commonly recorded in the pre-operative diagnoses. If a serous or sanguineous vaginal discharge cannot be accounted for otherwise, a diagnostic curettement should be done; and, if microscopic examination of the uterine scrappings fails to reveal the source of bleeding, a cancer of the fallopian tube should be considered. Chronic salpingitis, hydrosalpinx or pyosalpinx, and tubo-ovarian cyst or abscess are the terms most frequently used to describe the condition at operation. In many instances the cancer would probably have been recognized, or at least suspected, at the time of operation if upon its removal the tumor had been immediately opened and inspected.

TREATMENT

Failure to recognize the disease has resulted in conservative operations in a majority of cases. Upon receipt of the pathological diagnosis, a second operation for the removal of the remaining

internal genitalia has occasionally been done. In many patients the condition was too far advanced for anything but palliative measures. Complete extirpation of the uterus and adnexa via the abdominal route is generally advocated. Some authors believe it is unnecessary to remove the cervix as metastases are rarely found there. Bower and Clark would preserve the cervix for subsequent radium implantation. Excision of all palpable pelvic and retroperitoneal lymph nodes has been advised. This procedure is questionable, since metastasis to the intricate pelvic lymph system permits little hope for a surgical cure. Postoperative, deep roentgen-ray therapy is frequently suggested and is undoubtedly worth a thorough trial. Follow-up records are too inadequate for the drawing of conclusions concerning the best method of therapy in these cases. Available statistics show that the radical removal of the uterus and adnexa has accomplished little, if any, improvement in the prognosis of the disease. Nevertheless, the fact that the disease is bilateral in about one-third of the cases plus the fact that the ovaries and the uterus, though macroscopically normal, may be implanted with the disease are sufficient indications for the radical procedure, despite its apparent failure to alter the prognosis.

PROGNOSIS

Based on follow-up records, the operative results are found to be extremely poor. Only 6 patients have survived the third postoperative year without a recurrence. Four of these have passed the 5 year mark without a recurrence.

The case reported below is interesting for the following reasons; (1) it is the only case of primary carcinoma of the fallopian tube on record in the department of pathology at the University of Oregon Medical School; (2) it is one of a minority group of these cancers in which there is no evidence of previous pelvic inflammation; (3) the nearest approach to a characteristic symptom (watery vaginal discharge) was present and exaggerated; (4) the disease was recognized at operation and treated radically; and (5) the disease was confined to the outer half of one fallopian tube and subjacent ovary with no evidence of further extension or metastasis.

REPORT OF CASE

Mrs. C. S., white, aged 50 years, housewife and laundry-worker, entered the Multnomah County Hospital, November 8, 1928, complaining of watery vaginal discharge. Patient's mother had died of "stomach trouble" at the age of 61 years. A sister had died following an abdominal operation for "tumor" at the age of 52. Patient had had no severe illnesses and no operations. She had had ten full term pregnancies without complication. There had been no miscarriages or abortions. The menstrual cycle, always normal in every way, had ceased abruptly with the last period in October, 1924. During the past 18 months symptoms characteristic of the climacteric had appeared.

The onset of the present illness was 7 months ago, was marked by the sudden appearance of a blood tinged, watery discharge, which stained her clothing. Previous to this she had had no vaginal discharge. The discharge, continuous from the first, had become gradually more profuse and 6 weeks after the onset had lost its staining qualities. During the past 6 weeks napkins (each composed of a folded hundred-pound flour sack) had to be changed three or four times during the day and twice during the night. During and following exertion there had been a mild, dull aching pain in the lower quadrants of the abdomen which in the last 6 weeks had become more severe. Sacral backache and sharp shooting pains in the lower abdomen had produced uncomfortable nights for over 2 weeks. She had been able to keep up her work, but had suffered from pain and fatigue. Her appetite had been poor for 1 month and she felt that she had lost weight and strength. Slight soreness in the lower right quadrant of the abdomen had been present for 1 week.

Physical examination disclosed firm, moderately movable, tender mass about 6 centimeters in diameter posterior and to the right of the uterus. This mass was slightly adherent in the region of the right utero-sacral ligament. The laboratory reports were irrelevant.

Provisional diagnosis: (1) tubo-ovarian mass, probably inflammatory in character; (2) cancer of the fundus uteri.

A diagnostic curettement revealed nothing suspicious of cancer. The abdomen was opened through a low midline incision. There were no signs of inflammation and no free fluid in the abdomen. The uterus and left adnexa appeared normal. The right fallopian tube was enlarged in its distal half to form an oval tumor about 5 by 6 centimeters in diameter. This was slightly adherent and easily separated from the posterior parietal peritoneum in the region of the right uterosacral ligament. A small, thin walled cyst, not connected with the ovary, was attached to the closed fimbriated extremity of the tube. The cyst ruptured on manipulation and released a small amount of clear serous fluid. The right ovary showed no gross abnormalities and was not connected with the tubal mass. The right adnexa, including the previously mentioned cyst, were removed with a wedge of uterine muscle. The tubal mass was opened and revealed a hæmorrhagic, friable, papillomatous growth which had the appearance of cancer. In view of this finding the entire uterus with the left adnexa was completely removed. No pelvic lymph nodes were felt.

The patient was discharged in excellent condition after a normal convalescence of 14 days. She was advised to return at regular intervals for examination and deep roentgen-ray therapy. Hospital records show that on December 14, 1928, she received 250 milliampere minutes of X-ray, front and back.

March 6, 1929, 5 months after operation the patient was seen by the writers. She had no complaints and the pelvic examination was irrelevant.

Pathology. The uterus and left adnexa appeared normal. The proximal half of the right tube was normal except for a slight increase in its diameter, whereas, the distal half was greatly enlarged to form an oval tumor measuring 3 by 4 by 5 centimeters. The fimbriated extremity of the right tube was closed save for a small cyst not connected with the ovary. An opening in the cyst revealed a smooth interior surface which embraced one of the tubal fimbria. The serosa of the tube was pale pink, smooth and glistening, except for a few small roughened areas due to adhesions. An

opening in the thickened wall of the distal half of the tube revealed a hæmorrhagic, friable, papillomatous growth which occupied the entire lumen (Fig. 1). When sectioned the proximal half of the tube was found to be patent with no gross evidence of pathological change. The right ovary measured 2.5 by 3.5 centimeters and had a bossed surface which was due to a few small superficial cysts plus several firm, elastic nodules. Bisection of the ovary revealed a grayish surface with several small encapsulated compact areas which were non-friable.

Sections from the uterus and left adnexa appeared essentially normal. The proximal third of the right tube was normal save for a slight increase in the diameter of its lumen. Sections from the middle third of the right tube 1 centimeter from the tumor showed a slight round cell infiltration and a slight hypertrophy of a few of the mucosal folds. Sections from the tubal wall, which encapsulated the growth, revealed a patchy, round cell infiltration of all the coats with a definite increase in connective tissue elements and a marked distortion and a hypertrophy of the mucosa. There were few normal mucosal folds. The majority of these were composed of thick, aborescent, fibrous tissue cores covered with single and multiple layers of epithelial cells (Figs. 2 and 4). Where not distorted by crowding the tumor cells were low columnar or cuboidal in type with cytoplasm that took a light eosin stain and nuclei that were irregular in size, shape, and staining reaction (Fig. 3). A few mitotic figures were seen (Fig. 5). In many areas alveolus-like masses of cells appeared to have sprung from the mucosa between the papillary folds and to have invaded the wall as well as the lumen. The serosa of the tube was not penetrated by tumor cells. The dense, friable, central portion of the growth was composed of papillary chains of cells which in areas were so closely placed that the original papillary pattern was almost indistinguishable. In addition there were areas in the tumor without particular pattern which were composed of closely packed, large, polygonal cells with clear, faintly staining cytoplasm and nuclei. Many of these cells were vacuolated and partially disintegrated. The previously noted ovarian nodules had a fibrous capsule which surrounded compact cellular masses. The latter were composed of round, oval, and spindle-shaped cells with a faintly staining cytoplasm and irregularly shaped nuclei that took a light hæmatoxylin stain. Hyperchromatosis and mitosis were less common than in the tumor cells of the tube. A few corpora fibrosa were seen. The greater portion of the ovarian stroma was replaced by the tumor nodules. Invasion of the ovarian capsule by the tumor cells was not noted.

CONCLUSIONS

1. Primary carcinoma of the fallopian tube is a rare disease. About 200 authentic cases are recorded in the literature.

2. Age incidence is the same as for cancer in general.

3. The etiology is unknown, though many authors are of the opinion that chronic salpingitis, commonly associated with the disease, is a predisposing factor.

4. There are no characteristic physical signs or symptoms. Watery vaginal discharge, blood-tinged at times, is the nearest approach to a characteristic symptom.

5. The diagnosis has been made only once (by Falk) before operation. In a large percentage of the cases the disease was neither recognized nor suspected at operation. The case reported demonstrated the value of immediate section and inspection of extirpated pelvic tumors.

6. Statistics are too inadequate for the drawing of conclusions concerning the best method of therapy. The very nature of the disease, however, indicates a radical extirpation of the uterus and adnexa.

7. The prognosis is poor. Few cases have survived the fifth postoperative year.

8. Macroscopically, the disease resembles a chronic inflammatory condition (often indistinguishable from a pyosalpinx or hydrosalpinx) of one or both tubes. On opening the tube a friable, papillomatous growth is commonly noted.

9. Microscopically, the essential picture is a papillary or papillary-alveolar type of tumor. The primary disturbance appears to be a malignant hyperplastic change in the cylindrical epithelium of the tube.

10. The subjacent ovary is frequently involved.

11. Retroperitoneal lymph gland metastases are common.

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PATHOLOGICAL FRACTURES

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A PATHOLOGICAL fracture may be construed as any fracture occurring through a demonstrable bone lesion and implies a causative stress too trivial to produce the same result in normal bone.

In a review of 1,405 cases of fracture of the long bones at the Massachusetts General Hospital during the 10 year period from January, 1910, to January, 1920, there were 24 cases of pathological fracture. Fractures of the type of Potts' and Colles' and those fractures of the clavicle treated as ambulatory cases were not included in the cases reviewed. Of the 24 cases obtained 14 were females and 10 were males, and the ages included ranged from 9 months to 66 years. Table I shows the distribution as to age and sex of these cases and it is interesting to note that of all the long bones the greatest number of pathological fractures involved the femur where, of 560 cases of fracture, 15 were spontaneous and of these the fracture was below the trochanters in 9 instances. One case of fragilitas ossium, not shown on the chart, was a female baby 9 months old with multiple fractures involving all the long bones except the right humerus and clavicle.

The abnormal bone conditions responsible for these fractures and the bones involved are listed in Table II and it is immediately apparent that Paget's disease and osteomyelitis accounted for almost half the number of fractures.

The patient with hypernephroma and the 2 with metastatic carcinoma died in the hospital from the original condition, while the case of gumma was a woman, aged 42, who had had a slightly tender lump over the clavicle for a year and an X-ray picture taken after admission to the hospital showed a fracture of the clavicle, but the patient did not know when it had occurred. Noon reports 2 cases of gumma causing pathological fracture in a series of 5 cases which, however, were all in soldiers. In the three cases of sarcoma, one occurred in a girl of 9 years who fractured the lower end of the right femur in a fall. A high amputation was done and the pathological report showed large spindle cells and giant cells and the diagnosis of sarcoma, made before operation, was confirmed. Four years later she was well, and there was no evidence of further involvement. The 2 other cases were adults, ages 53 and 60, and the outcome is unknown as the patients left the hospital untreated. The diagnosis in these cases was based on the X-ray findings and may be erroneous. Moore calls attention to

TABLE I.—DISTRIBUTION AS TO AGE AND SEX

		Femur	Tibia	Fibula	Humerus	Radius and Ulna	Clavicle	Total
Number of fractures		560	142	97	313	208	85	1405
Pathological fractures		15	4	0	3	0	1	23
Sex	Male	7	3	..	..	..	..	10
	Female	8	1	..	3	..	1	13
Age	Male	17 to 66	52 to 63	..	..	..	..	..
	Female	2 to 53	20	..	11 to 63	..	42	..

TABLE II.—BONES INVOLVED

	Male	Female	Femur	Tibia	Humerus	Clavicle	Total Cases
Paget's disease	4	2	4	2	0	0	6
Osteomyelitis	3	2	4	1	0	0	5
Cyst	0	3	1	1	1	0	3
Sarcoma	0	3	3	0	0	0	3
Fragilitas ossium	0	2	1	0	0	0	2
Metastatic carcinoma	2	0	1	0	1	0	2
Syphilis	1	0	1	0	0	0	1
Gumma	0	1	0	0	0	1	1
Hypernephroma	0	1	0	0	1	0	1
Total	10	14	15	4	3	1	24

his experience that bony metastases of all types of malignancy give the same roentgen appearance, thus emphasizing the fallacy of attempting a cellular diagnosis by the roentgenogram.

In 2 of the 3 cases of bone cyst, the patient had had a previous spontaneous fracture in another bone due to cyst and in each instance the fracture was the first indication of the presence of abnormal bone.

In this series of cases the most common causes of pathological fracture were Paget's disease and osteomyelitis with sarcoma and cystic disease next in rank. It is interesting in this regard to compare Eisendrath's report of the Cook County Hospital where tabes was most frequently encountered, which led him to advise an examination of the nervous system in all cases of fracture in which manipulation causes little pain.

The average age in these 24 cases was 38.1 years compared to the average age given by Meyerding of 26.7 years. Nine males were over 30 years of age and 5 females were over 30 years of age.

In Rassieur's series of 588 fractures, 5 were pathological and were caused by syphilis or sarcoma. He concluded that spontaneous fracture was usually a sign of impending death.

Very frequently the spontaneous fracture was the first warning that disease of the bone was present and according to Boggs this may also be true in cases of bone metastases. Nevertheless, the fracture, as Ashhurst says, "is a complication and does not alter the prognosis or the indications for treatment." Bone graft is not necessary to secure union in non-malignant conditions: curettage with proper postoperative fixation may be quite sufficient (12) as non-union is not usual after pathological fracture (5). Bands used in the fixation of fractures have been responsible for spontaneous fracture at the site of the band in the experience of one writer (6). In cases of malignancy of the bone, whether primary or metastatic, union rarely takes place and death often intervenes before any reparative process begins. This view is supported by many writers and has been tersely set forth by Codman. However, spontaneous fracture due to metastatic carcinoma sometimes unites quickly although the patient succumbs to the malignant disease (9).

A review of the literature on pathological fracture revealed only 18 cases of fracture due to echinococcus cyst, and a new hazard was brought to light as a cause of bone pathology in the use of radium paint by watch dial painters. One girl thus employed had a spontaneous fracture through the upper third of the femur producing extensive crippling (7).

Of the 24 cases of pathological fracture obtained in this series, 3 died in the hospital, 6 were untreated, and 15 were well from 1 to 4 years after leaving the hospital.

CONCLUSIONS

Pathological fracture is a rather common incident to bone pathology and frequently is the first indication of its presence.

Pathological fracture in non-malignant conditions does not make the prognosis unfavorable.

Indications for treatment of the underlying condition which results in fracture are not altered by the fracture.

Cellular diagnosis of bone malignancy by means of the X-ray may be erroneous.

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INDIRECT INGUINAL HERNIA

Some Observations on Russell's Theory and Technique

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RUSSELL, in 1925, set surgeons in this country to thinking on the subject of the repair of indirect inguinal hernia. Many did not, and have not to this day, accepted his theory, although his reasoning and conclusions seemed logical and clear. Some men have accepted the theory and are repairing indirect inguinal herniæ by the method suggested by him, but with certain modifications. Counsellor, of the Mayo Clinic, for instance, obliterates the sac by twisting, but he fixes the stump by suturing through the muscle layers anterior to the internal ring. He leaves the muscles of the falx inguinalis intact, however, as outlined by Russell.

A case, which came to autopsy 6 hours after operation, in which the classical procedure described by Russell was followed, gave us the opportunity of observing just what happens with the stump of the sac.

It is our purpose in this article to show that, if properly done, this operation leaves the parietal peritoneum at the abdominal inguinal (internal inguinal) ring in better condition, so far as prevention of herniation is concerned if one would allow that this was possible, than in a normal abdomen in which complete closure of the tunica vaginalis has occurred at birth. We purpose, also, to sustain Russell's contention by giving an example of a case, which argues in favor of the preformed sac theory. The history of this case will be recognized by all surgeons as the counterpart, in its main aspects, of nearly all cases of indirect, inguinal hernia. We are speaking of indirect hernia and, not in any sense, of direct hernia. This should be kept in mind, because the cause and effect and cure of the two are often confused in the discussion of hernia.

Consider the cases of two newborn infants of equal robustness, with equally well developed skeletal musculature, one with an indirect hernia and the other with no hernia. The abnormality in the former is due to a patent tunica vaginalis. The muscles of the inguinal region are the same in both, and the rings of the canal are of the same size in both. The hernia is there then, or will appear, not because of a muscle defect but because of the presence of a sac lined with glistening endothelium which is ready to receive any of the abdominal contents that may find their way into it. If, in any way, we are able, in the abdomen with the hernia, to approximate or even better the picture presented at the abdominal inguinal ring of the well infant, then we should be able to expect that the child with the repaired hernia has the same chance of going through life free from an indirect hernia as the other child who has never had a hernia. It has never had one because it has never had a patent tunica. The muscles of the region have not entered into the picture, and it is reasonable to expect, as Russell suggests, that any attempt at their repair is not only wasted effort but may result in actual weakening of the wall because of strangulation of tissue with sutures, and subsequent atrophy, which may lead to a direct hernia. This latter is often spoken of as a recurrence, which it is not.

In the adult with an indirect hernia, the same conditions prevail as in the infant. The potentialities of hernia have been present from birth. A case in point, which is the counterpart of many such cases, is briefly reported.

A man, 38 years old, was driving an automobile. He was forced off the road, and his car hit a culvert. This drove his abdomen very forcibly against the steering wheel. A few days later, I was asked to examine him completely, because of the possibility of future litigation. He complained of a soreness across the lower abdomen. Examination of the right inguinal region showed a slight impulse against the tip of the examining finger, when the patient coughed. A small hernia was evidently present, but what is most interesting is that in a matter of 3 weeks the hernia was apparent in the upper part of the scrotum. This, despite the fact that he was taking things easy, resting most of the time. It is much easier to believe that a preformed sac was present, which was waiting to receive abdominal contents at any time that pressure applied above, would force those contents into the sac than that the peritoneum could pouch and stretch downward to the scrotum in 3 weeks because of a weak muscle at the internal ring(3). A repair by Russell's method then, would leave the muscles as they had been during this individual's life up to the time of the accident, but would obliterate any dimpling of peritoneum at the internal ring.

If, then, we can so repair the peritoneum that it is the counterpart of that lining a normal wall, we need not be concerned with the various types of muscle plastic work on the inguinal canal. We surely often see a lax, weak, abdominal wall in a poorly nourished individual, which is not the site of an indirect inguinal hernia. Why, then, do we

continue to disturb the normal relations and nourishment of the wall of an inguinal canal of a robust individual with well developed muscles? Here, again, we will say that a direct hernia is a different proposition—something that must always be looked for when a repair of an indirect hernia is being done. One can conceive of a long standing, large hernia of the scrotal type, which has stretched the muscles of the inguinal region to such a thin, atrophic state, that more than Russell's technique must be applied. In these cases an operation with fascial sutures, after Galli, would be indicated.

The autopsy finding in the case, which follows, proves beyond doubt that the parietal peritoneum about the internal ring, following Russell's technique, will not only be free of any indentation pointing into the inguinal canal but will actually show a cone of tissue pointing superiorly.

In May, 1926, a man, 47 years old, was admitted to the hospital for the repair of a right, indirect, inguinal hernia. The swelling in the inguinal region was first noticed after he had lifted an unusually heavy mail bag. Russell's technique was followed in the repair. The hernia was of 2 months' standing. The man was a hard drinker and, unknown to his physicians, had been imbibing for several days before his entrance to the hospital. He was given an ethylene anæsthesia. He was very obstreperous while being taken to his room and became quite violent and unruly in his bed. Opiates did not control this violence. He then went into a stupor, such as is seen in alcoholics following violent delirium tremens. He died 4 hours after the operation. At autopsy the pathologists were unable to find a cause for the death.

The autopsy gave us the opportunity of examining, within a few hours, the appearance of the peritoneum, which had been crushed and tied at the base of the hernial sac. We expected to find a slight dimpling downward into the inguinal canal; but, to our surprise, there was an actual "upward coning" of the stump. The left side, at the internal ring, showed a normal closure of the tunica vaginalis. This "upward coning" occurs, undoubtedly, as a result of the elastic recoil of the peritoneum, when the stump of the twisted sac is severed.

It seems to us then, if this is what happens—and it does, if all preperitoneal fat is well cleaned from the sac and the sac twisted, until there is an elastic pull from the peritoneum above, before the neck is crushed, tied, and cut—it is needless to transplant the stump into the anterior abdominal wall. In fact, it would seem that this procedure merely prevents the "upwarding coning" of the stump. This "upwarding coning" seems to us to be a desirability. It surely eliminates all dimpling toward the canal.

It has been easy for us (6) to follow the procedure, outlined by Russell, in children, but it is surely difficult to break away from the older methods in adults. We have had no recurrences in infants or adults in this type of operation. Counsellor has followed the method for the past year with good results. In a recent personal communication (1) one of our prominent surgeons in hernia work states that he is ready to try Russell's technique in a large series of cases.

CONCLUSIONS

1. When properly done, the peritoneum at the internal inguinal ring presents an "upwarding coning," thus eliminating completely the entire hernial sac.

2. Elimination of the sac is the thing for which the surgeon should strive.

3. Muscle transplanting operations are not necessary in indirect, inguinal herniæ of recent origin or of moderate size in adults.

4. Russell's technique is ideal in infants with indirect inguinal hernia.

5. Careful study of the clinical history and physical findings in cases of indirect inguinal hernia, tend to prove the theory of a preformed sac as the potential cause of herniæ.

6. The interesting details of Russell's technique should be carefully studied, in his articles, before attempting the operation.

7. An examination for the presence of a direct hernia must always be made, when a repair of an indirect hernia is being done. Repair of such a defect will eliminate many so-called "recurrences."

8. When there is practical destruction of the walls of the inguinal canal because of the presence of a long standing, large, indirect hernia, an operation of the Galli type is indicated (4).

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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JULY, 1930

EVALUATION OF STATISTICS

THE use of statistics in support of methods of treatment of disease is essential. Statistics should represent facts, but occasionally they do not, because of incomplete or insufficient data, omission of data not in agreement with the argument, or unjustifiable conclusions. These faults are avoidable. The difficulty of compiling accurate statistics often is due to inherent difficulties in ascertaining the truth, and it is for this reason chiefly that statistical studies of the results of therapeutic measures frequently are so confusing that the reader finds himself confronted with the necessity of evaluating the statistics rather than the questions they are supposed to solve.

In no field of surgery are these facts concerning statistics more apparent than in the benign lesions of the stomach and duodenum. The difficulty of estimating the effectiveness of surgical treatment of any lesion that is characterized by dyspepsia is obvious, and the ease with which the unfavorable results of any specific method may be assembled is often too readily made use of by advocates of some other method. Any therapeutic principle must rest on its merits rather than on the demerits of other principles of treatment. The practice of emphasizing the failures which may follow any surgical treatment for peptic ulcer in an attempt to substantiate one treatment for all cases has tended to submerge the fundamental principle in the surgical treatment of peptic ulcer; namely, the selection of the best method to be followed in the individual case.

It is refreshing, therefore, to find in a collective investigation under the direction of Arthur P. Luff and under the auspices of the British Medical Association[1] a compilation of statistics on the results of the surgical treatment of duodenal ulcer which is convincing because it is a sincere effort to assemble facts rather than opinions. It is particularly convincing since it is a report of a large number of cases in which operation has been done by 86 surgeons in Great Britain. The fact that the results are not the experience of an individual is most significant and should be remembered when comparison is made with the results reported by those advocating particular methods.

The outstanding facts included in this report are concerned with those points about which there has been much debate. The first is that in about 1,500 cases of lesions of the duodenum or of lesions encroaching on the pylorus from the duodenum, and in which operation was performed, an indirect operation, gastro-enterostomy, was used in more

[1]Luff, A. P. The after-history of gastro-enterostomy. Part I. Brit. M. J., 1929, ii, 1074–1078. Part II. Brit. M. J., 1929, ii, 1125–1129. Editorial. End-results of operation for gastric and duodenal ulcer. Brit. M. J., 1929, ii, 1066–1067.

than 95 per cent, and partial gastrectomy in about 2 per cent. British surgeons, with few exceptions, advocate conservative surgical procedures in duodenal lesions, and this report, considered from any angle, amply justifies the practice. The report shows, for example, that in 90 per cent of the cases in which gastro-enterostomy was done, satisfactory results were obtained; the average time at which the results were evaluated was 7 years after operation. Gastrojejunal ulceration occurred in 2.7 per cent of the cases in which the lesions were classified as duodenal, and in 0.9 per cent of those in which they were classified as pyloric. Subsequent hæmorrhage was reported in 2.4 per cent in the group of duodenal lesions and in 1.2 per cent in the group of pyloric lesions. As might be expected, carcinoma did not develop in any of the cases of duodenal lesions, and since there was none in the group of pyloric ulcers the fact is substantiated that the lesions in the latter group were primarily duodenal. Experience has shown conclusively that lesions which extend on the gastric side of the pylorus may be carcinomatous at the time of operation or they may later become malignant, and that, following indirect procedures alone in a sufficient number of such cases, death from carcinoma will occur in a certain number of instances.

All in all, the report is most impressive in its simplicity and in its adherence to facts, and it should do much to remove any doubt from the minds of those who may still be uncertain as to the value of adopting conservative measures in the surgical treatment of cases of duodenal ulcer. National medical associations may find in the report a suggestion of value as to the carrying out of similar investigations in the same impartial way.

DONALD C. BALFOUR.

RECURRENT AND PERSISTENT HYPERTHYROIDISM

IT is now generally accepted that the removal of sufficient hyperplastic thyroid tissue will relieve a patient of hyperthyroidism. If, however, there is a recurrence or a persistence of the hyperthyroidism after thyroidectomy, there is a very general tendency to postpone too long any secondary operation upon the thyroid gland.

Failure to relieve so called thyroid toxicity by thyroidectomy may be due to the fact that hyperthyroidism was never present and the operation was performed with an erroneous diagnosis. Such a case must be distinguished carefully from true persistent hyperthyroidism and every effort made to treat the real cause of the symptoms. When, in questionable hyperthyroidism, the primary removal of a large amount of thyroid tissue fails to produce marked relief of symptoms, the diagnosis of hyperthyroidism should be strongly suspected of being wrong. Probably true hyperthyroidism was never present and further surgery should be either long delayed or entirely avoided.

True recurrence of hyperthyroidism occurs when, after an interval of twelve months or more with a normal basal metabolic rate and freedom from symptoms following thyroidectomy, the basal metabolic rate is again elevated and symptoms of thyroid toxicity reappear. True recurrences are quite rare but do occasionally occur.

Persistent hyperthyroidism, on the other hand, is said to be present after thyroidectomy if the basal metabolic rate fails to return to normal and the symptoms of hyperthyroidism never entirely disappear. Persistent hyperthyroidism is not uncommon and is the usual finding in cases of exophthalmic goiter not relieved by operation.

Recurrent hyperthyroidism should be treated as though no previous operation upon the thyroid had been performed. After proper preparation with rest and Lugol's solution, secondary thyroidectomy, with the thorough removal of all excess hyperplastic tissue, is undertaken. These measures will bring relief in the vast majority of cases. The possibility of further recurrence is slight.

In persistent hyperthyroidism, however, when the basal metabolism has failed to return completely to normal after thyroidectomy and symptoms of toxicity persist, palliative measures may for a time be adopted. In certain patients, limitation of activities and the daily use of Lugol's solution will permit so comfortable and safe an existence that surgery can be, at least temporarily, delayed. Usually, however, these measures fail and when after a fair trial the basal metabolism remains elevated and complete relief of the hyperthyroidism is not obtained immediate secondary thyroidectomy should be advised without further delay. Prolonged temporizing with definite persistent hyperthyroidism should be avoided just as rigidly as in primary hyperthyroidism since long standing hyperthyroidism carries with it so many undesirable complications. Secondary thyroidectomy in persistent hyperthyroidism cases gives most satisfactory results when it removes completely the excess hyperplastic tissue. It should be urged when it is obvious that the primary operation has failed to relieve the patient of hyperthyroidism.

HOWARD M. CLUTE.

MASTER SURGEONS OF AMERICA

FRANCIS JOHN SHEPHERD

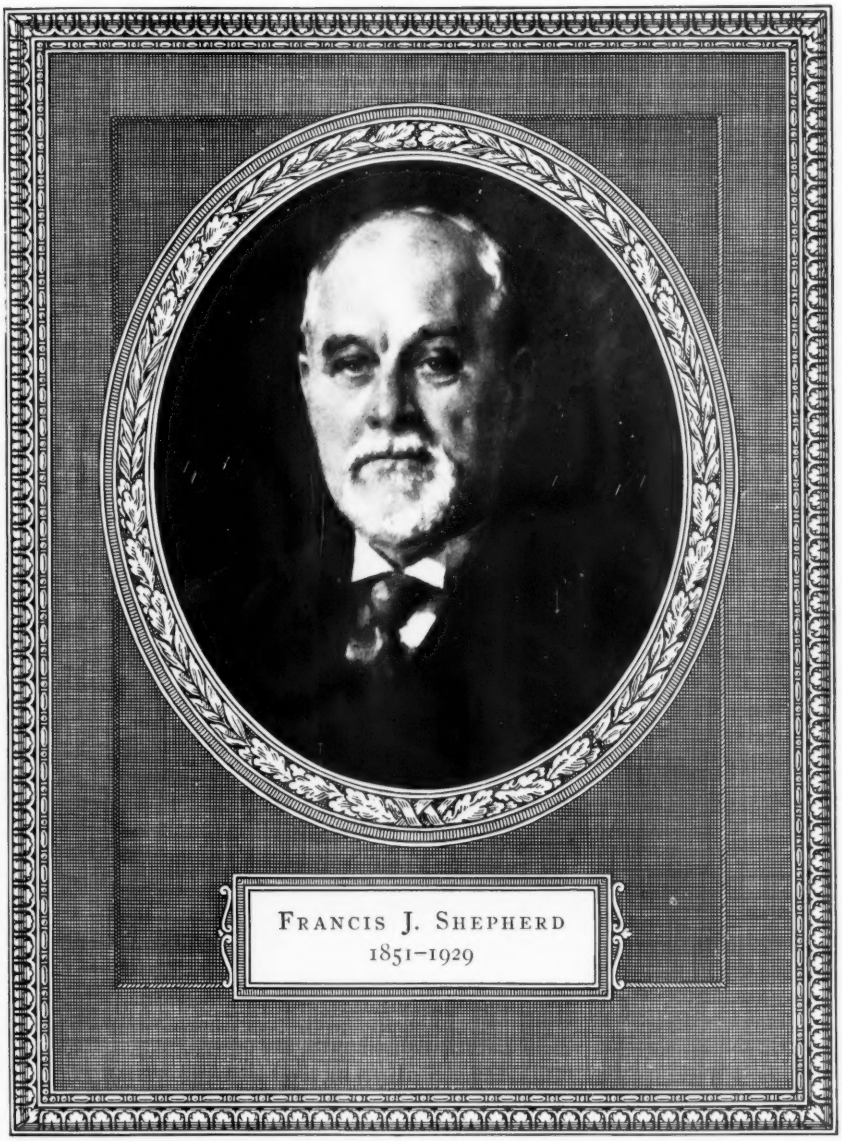
IN Vaudreuil, near Montreal, Francis John Shepherd was born in 1851. His early education was received in local schools, and after spending a year in the Arts Faculty he entered the Faculty of Medicine, McGill University, and graduated in 1873. He then went across the Atlantic for two years' further study in the hospitals of London, Paris, and Vienna, and he was asked by McGill University to qualify as a teacher of Anatomy. This he did in his usually thorough manner. In Vienna he was joined by two distinguished graduates of McGill, George Ross, afterward professor of medicine in McGill, and William Osler, who graduated in 1872.

On his return to Montreal he was given the position of demonstrator of anatomy in the University and on the death of Professor Scott was made full professor. He established at McGill a school of human and comparative anatomy of a very high standard, recognized as such in America and in England, so that the students of McGill received a very thorough training in scientific and applied anatomy. In this work Dr. Shepherd associated with himself a number of promising young men who afterward attained enviable positions in medicine, surgery, and the specialties. After twenty-five years' service as professor of anatomy he was tendered a banquet by his past and present assistants on the teaching staff and by them given a fine silver coffee service.

Dr. Shepherd's interest in medical students was not confined to teaching them anatomy. Together with Ross and Osler, he founded an undergraduate society of medical students with regular meetings for the discussion of interesting questions.

He was always interested in the development of the pathological museum, particularly the anatomical anomalies, of which he had accumulated a large and rare collection. Unfortunately, this collection was lost in a fire which destroyed the building in which they were housed.

The Montreal Medico-Chirurgical Society enjoyed a period of unusual prosperity under the stimulating influence of Osler, Ross, and Shepherd. For many years he was one of the editors of the *Montreal Medical Journal* and contributed a surgical retrospect for each number that was critical, instructive, and interesting. These retrospects were afterward published in book form.



FRANCIS J. SHEPHERD
1851–1929

In 1883, he was elected full surgeon to the Montreal General Hospital and organized a course of summer clinics for students who remained in Montreal during the summer months. These clinics were very popular and much appreciated.

While in Vienna, Dr. Shepherd took a special course in dermatology under Hebra, and on his return started a department of dermatology in the Montreal General Hospital. His interest in the subject continued to the end. In 1907 he was named vice-president of the Dermatological Congress held in New York and in 1928 was made president of the Canadian Branch of the British Dermatological Society.

Dr. Shepherd's surgical work merits the highest praise. He was a most careful diagnostician, and his operative work was bold and progressive yet at the same time conservative. He was particularly interested in the development of the surgery of the thyroid. I was intimately associated with him for many years and always found his opinion sound. Under his teaching in the old summer sessions many young men received a training in surgery at the beginning of their career that left an impress that was never lost. Always careful, the interest of his patient was ever foremost in his mind.

Dr. Shepherd's work in anatomy, dermatology, and surgery was appreciated outside his native land. In 1913, he was chosen to act as vice-president of the department of surgery at the International Medical Congress in London. He received an honorary fellowship from the Royal College of Surgeons, Edinburgh, and had the degree of doctor of laws conferred upon him by the University. He also received the much prized honor of the honorary fellowship of the Royal College of Surgeons of England. The degree of doctor of laws was conferred upon him by Harvard University in 1906, by McGill in 1915, and by Queens in 1919. He was also made an honorary fellow of the American College of Surgeons. He was elected corresponding member of the Sociéte Internationale de Chirurgie of Paris, and foreign member of the American Academy of Arts and Sciences.

In 1924, his numerous friends in the profession presented him with a portrait of himself, by Miss Des Clayes. This now hangs in the large hall of the medical building of McGill University along with the portraits of previous deans. He was dean of the Medical Faculty 1908–1914, president of the Medico-Chirurgical Society in 1894, and president of the Canadian Medical Association in 1901.

Dr. Shepherd contributed liberally to medical literature. In addition to his *Surgical Retrospect* already mentioned, he was joint author of *American Textbook of Surgery*, author of the article "The Thyroid" in the *American Practice of Surgery*. He contributed to the *Annals of Surgery*, *Journal of Anatomy and Physiology*, *Canadian Medical and Surgical Journal*, *British Medical Journal*, and *Lancet*.

Dr. Shepherd had many outside interests. He was fond of nature and fishing, and had a lovely country home at Como on the Ottawa river where he entertained

his friends in royal fashion. He was particularly fond of art, especially of oil painting, and indeed was regarded as an authoritative critic. He was president of the Montreal Art Association from 1906 to 1910, and in 1918 he was again persuaded to take that position which he was still occupying at the time of his death. On the occasion of his seventy-seventh birthday, he was presented with a bronze bust of himself by a circle of his friends in the association.

For many years he was a member of the Board of Trustees of the National Gallery at Ottawa and recently chairman of the board. He was keenly alive to the value to the country of collections of fine art, not only for educational purposes but for the increase of national pride in art and a stimulus to all who may be able to enrich their country with further valuable examples.

The public will remember Dr. Shepherd's achievements; his patients will remember him as a kindly and skilled physician and surgeon; his associates and friends will cherish the remembrance of his character. He was frank, outspoken, versatile, and a man of unimpeachable integrity. Dr. Shepherd was a widower for many years. He had one son, whom he lost in the war, and two daughters—Mrs. Nobbs, and Miss Dorothy—who devoted her life to the care of her father.

On January 17, 1929, he was at his club and in a particularly happy mood. The following morning when his breakfast was taken in to his room he seemed cheerful as usual. When a little later the servant returned to take away the tray, she found him sitting up in bed, his spectacles on, the morning paper in his hand, but he had passed on.

GEORGE E. ARMSTRONG.

ZACVTI LVSITANI,

Medici, & Philoſophi præſtantiſſimi,

OPERVM TOMVS PRIMVS,

IN QVO

DE MEDICORVM PRINCIPVM HISTORIA.

LIBRI SEX.

Vbi Medicinales omnes Hiſtoriæ de morbis internis, quæ paſſim apud Principes Medicos occurrunt, concinno ordine diſponuntur, Paraphraſi, & Commentariis illuſtrantur: necnon Quæſtionibus, Dubiis, & Obſeruationibus exquiſitiſſimis exornantur.

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THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

ALFRED BROWN, M.D., F.A.C.S., OMAHA, NEBRASKA

THE COLLECTED WORKS OF ZACUTUS LUSITANUS

ABRAHAM ZACUTUS, more commonly known as Zacutus Lusitanus, was a Portuguese Jew who was born at Lisbon in the year 1575. The descendant of an old Jewish family, he was given the opportunity to obtain an excellent education. He studied at the Universities of Salamanca and Coimbra and not until 19 years later at Seguenza, a town in the province of Segovia, was he given his doctor's degree. He practiced in Lisbon for 30 years and after building up a magnificent practice and making an excellent reputation, he suffered with the rest of the Jews in Portugal, from the order of banishment which was proclaimed in 1625 by King Philip IV. Zacutus left Portugal and went to Amsterdam where he spent the rest of his life in practice and died in 1642. These facts are given in the history of his life which is contained in the first volume of his collected work, written by Ludovicus Lemosius of Lisbon who was physician in ordinary to the Royal Court.

Daremberg, writing in 1870, makes the statement concerning Zacutus' work that it is "a work which is still very useful." It seems to me that this same statement holds true today, and I do not believe that the book can be summed up better than it is summed up on the title page of the first volume, which says: "Where all the medical histories of internal disease which are met with here and there among the principal medical men are arranged in proper order and are illustrated with paraphrases and commentaries; and also are adorned most exquisitely with questions, criticisms, and observations." Zacutus has searched the literature up to his time, not neglecting the literature of his contemporaries, and taken all internal diseases, and some external, and arranged them in order from the head to the foot, most of his case histories being drawn from Galen and the Arabian physicians.

He arranges the matter in the following form. First comes the history of the case as it is given in Galen, or one of the Arabian authors. This is usually copied directly from the book as published. Then he takes up a discussion of the disease in question which he calls a paraphrase. Then, if he has had a similar case himself, he gives the history of his own case or one that he has seen with some other physician and gives the result of the treatment and where it may or may not differ from the idea of the old author. When speaking of his own case he usually refers to the ideas of his contemporaries. For instance, in his first observation concerning alopecia he says he cured a case of baldness with the juice of nicotiana which he says is vulgarly called tobacco. He then quotes a book, *The Book Concerning Tobacologia*, by Joannes Meander Bremanus. Where there are certain things of which he is doubtful he asks a rhetorical question and then discusses the question he has asked and usually decides in his own favor.

Zacutus, himself, apparently did not practice surgery, and when a case fell into the surgical class, called for a surgeon to do the operative work. Many of the cases scattered through the two volumes, however, are surgical and many of them are of exceeding interest. The forty-ninth observation of the third book is of extreme interest. Zacutus gives the history of a case which he entitles "A stone springing above the frenum of the tongue." He says that a certain noble was bothered with a catarrhal fluction which appeared in the external part of the tongue next to the frenum and caused a rather large painful tumor which almost made him lose his voice. A surgeon was called who washed it lightly with emollients and took council and then made an incision. A stone showed itself surrounded by yellow pus. It was hard, on the left side, and about the size of a filbert. It was then extracted with a vulsellum and the tumor disappeared. The wound healed safely and the patient was brought to perfect health. He then goes back in medical literature and quotes a case of stone in the tongue described by Avenzoar in his second book. Following this he gives a criticism and asks whether stones may form in various parts of the human body. This again is followed by a commentary, or discussion, regarding the formation of stones all over the body. Nearly all of the common surgical diseases are considered and many of the uncommon.

Zacutus had evidently read nearly all of the works of the ancient authors and also those of his more recent predecessors and even of his contemporaries. He shows himself to be an exceedingly clear and careful observer and, even though of necessity, his histories are reduced to small space and are not complete as we would consider completeness in a medical history today, nevertheless they are so plainly stated and the arguments so clearly carried out that in nearly every case it is possible to make a diagnosis of the condition found.

REVIEWS OF NEW BOOKS

VOLUMES III and IV continue the exhaustive German handbook[1] on urology. Like all such systems the quality of the chapters varies with the author. With one or two exceptions the classification is on clinical lines which results in some repetition which might have been avoided if the exposition had been based on pathological entities. In most of the articles extensive references to the literature are given, but as German authors do not have the assistance of our splendid medical indices of literature, they do not adhere to any special method of giving the references, with the result that not a few of them are incomplete and very difficult to find.

Anomalies are first considered. It is interesting to note that they have been divided into separate anatomical and clinical portions written by different authors. The section on malformations of the male sex organs is unusually good and contains much material hitherto unfamiliar to many. The clinical sections on malformations has a long study of the literature without the critical attitude one would hope for. The chapter on injuries contains much interesting war material. A chapter on diseases of micturition and one on nervous diseases of the bladder contains no mention of the splendid work of the English physiologists. To diffuse nephritis, a subject usually more or less excluded from American works on urology, 116 pages are devoted

The surgical treatment of nephritis and eclampsia is completely covered. It is especially interesting that neither Pleschner nor Voelcker recommends pyelography as an important diagnostic aid in renal carbuncle or renal tumor. Ureteral dilatation is not mentioned as a part of the treatment of renal infections. There is no reference to submucous fibrosis or Hunner ulcer of the bladder. In the treatment of gonorrhœa also no stress is laid on urethral dilatation, but it is mentioned only as one of the procedures with which to determine a cure. Renal tuberculosis is covered in an authoritative style by Wildbolz, but tuberculosis of the genital tract is done in much more academic fashion. Rosenstein presents an excellent monograph on actinomycosis.

The masterpiece of the two volumes, however, is the long chapter on renal stone by Gottstein. He covers every aspect of this subject in a masterly way, omitting only the latest studies on the effects of diet and vitamins on stone formation. Floersken makes some very sensible remarks concerning movable kidney. One of these deserves quotation, namely, "Never do nephropexy in cases of general enteroptosis or where there is a definite psychoneurosis." Hottinger in treating of bladder stone gives as much space to perineal lithotomy as to suprapubic. This is in contrast to the American practice. Voelcker and Boeminghaus used the term hypernephroid tumor instead of hypernephroma, which is certainly a step in the right direction. Curiously enough, echinococcus cyst is included in the chapter on solid tumors of the kidney.

The reviewer did not note any reference to the phenolsulphonephthalein test in the two volumes and only a few to the indigocarmine test. Apparently much stress is laid on the freezing point of the urine.

This work contains much of great value and it is extremely instructive as illustrating the practice of some of the masters of urology in Germany. The reviewer feels, however, that it would be a mistake for any American urologist to take it as his sole textbook.

DAVID M. DAVIS.

[1] HANDBUCH DER UROLOGIE. Edited by A. von Lichtenberg, F. Voelcker, H. Wildbolz. Vols. iii and iv. Special Urology, i and ii. Berlin: Julius Springer, 1927.

SIX years have elapsed since the first edition of Farr's excellent monograph[2] on local anæsthesia. The thoroughly revised second edition is much more than a lucid presentation of this subject. It is really a book on surgical technique admirably adapted to the requirements of local anæsthesia.

The psychic factors, the physical comfort, the posture on the operating table, a special injector, the wire spring retractors, and other ingenious devices, which had previously been described by the author all serve one purpose: smooth and successful teamwork and a surgical strategy, "without which even the most skillful 'nerve block' gives only half results." It may be argued, that the average surgeon can perform operations under local anæsthesia with less technical equipment, but the author's simple, practical methods of infiltration are easy to follow and are always successful.

One cannot help but admire a whole life's work as represented in this volume, which is dedicated to the comfort of that "unfortunate individual, the patient, who, fate has decreed, must undergo surgical treatment." The illustrations and the printing are excellent.

GEZA DE TAKATS.

[2] PRACTICAL LOCAL ANESTHESIA AND ITS SURGICAL TECHNIC. By Robert Emmett Farr, M.D., F.A.C.S. 2d ed. Philadelphia: Lea and Febiger, 1929.

A COMMITTEE appointed by the American Gynecological Society has prepared *A Syllabus of Lectures on Obstetrics for Nurses*[3] which is now ready for distribution to those interested in the subject of nurse training. Copies may be obtained from the chairman of the committee, at the nominal price of 50 cents by addressing a request to 23 East 93rd Street, New York, New York.

[3] A SYLLABUS OF LECTURES ON OBSTETRICS FOR NURSES.

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E. L. ELIASON, *Chairman* DRURY HINTON, *Secretary*

BROOKE M. ANSPACH
LOUIS H. CLERF
JOHN D. ELLIOTT
FLOYD E. KEENE
FIELDING O. LEWIS
GEORGE P. MULLER
WILLIAM J. MERRILL
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DAMON B. PFEIFFER
JOHN S. RODMAN
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B. A. THOMAS

PLANS FOR THE 1930 CLINICAL CONGRESS IN PHILADELPHIA

THE clinicians of Philadelphia are interested to provide a complete showing of the clinical surgical activities of that great medical center during the twentieth annual Clinical Congress of the American College of Surgeons to be held in that city October 13–17. They are preparing a program of clinics and demonstrations to be given in the hospitals and medical schools of that city that will include all branches of surgery—general surgery, gynecology, obstetrics, orthopedics, urology, and surgery of the eye, ear, nose and throat. Clinics are scheduled to begin at 2 o'clock on Monday, October 13, and for the mornings and afternoons of the following four days. A preliminary clinical program will be published in the August issue of this journal. Recalling the splendid programs presented at four previous sessions in this city one may confidently assume that this year's Congress will surpass all previous efforts in point of scientific interest.

A series of fracture clinics demonstrating modern methods in the treatment of fractures will form a special feature of the clinical program. At many of the larger hospitals plans are being made for a comprehensive showing of methods employed and results obtained in the treatment of fractures which forms so large a part of the surgical work in large cities and industrial centers.

A subcommittee composed of William T. Shoemaker, Chairman, J. A. Babbitt, Louis H. Clerf, Fielding O. Lewis and Luther Peter has been appointed to arrange programs for a series of clinical demonstrations on ophthalmological and otolaryngological subjects to be held at headquarters each morning except Monday, in view of the fact that the clinical work in these specialties will be demonstrated at the hospitals in the afternoons.

The Executive Committee of the Congress is preparing programs for a series of five evening meetings. On Monday evening at the Presidential Meeting the president-elect, Dr. C. Jeff Miller of New Orleans, will be inaugurated and will deliver the annual address. Another feature of that meeting will be the annual Murphy oration in surgery. Distinguished surgeons of the United States and Canada, and eminent surgeons from abroad have been invited to present papers dealing with surgical subjects of present day importance at scientific meetings on Tuesday, Wednesday and Thursday evenings. On Friday evening at the annual Convocation of the College, the 1930 class of candidates for fellowship in the College will be received.

An interesting feature of this year's Congress will be the showing of a number of surgical films that have been produced under the supervision of and approved by the Board on Medical Motion Picture Films. A number of these films have been completed since the Congress in Chicago last year and such films will be given their premier showing in Philadelphia. An extensive program, to include other outstanding contributions not comprised in the College library of films, is planned.

At the annual meeting of the College on Thursday afternoon, beginning at 2 o'clock, formal reports on the activities of the College will be

presented by the officers and several standing committees. The major portion of the afternoon will be devoted to a symposium on cancer, dealing with various aspects of this important problem.

In addition to the Executive Committee as listed above, the following representatives of hospitals and medical schools have been appointed as members of the Committee on Arrangements: E. G. Alexander, Leon T. Ashcraft, W. Wayne Babcock, William Bates, John A. Brooke, H. P. Brown, Ralph Butler, B. F. Buzby, G. M. Dorrance, L. D. Englerth, Ralph Goldsmith, J. Milton Griscom, Robert Kimbrough, Edward J. Klopp, A. D. Kurtz, Benjamin Lipschutz, Clifford Lull, P. A. McCarthy, J. J. A. McMullin, George M. Marshall, George H. Meeker, William E. Parke, William N. Parkinson, Ross V. Patterson, William Pepper, William Pierson, Warren Reese, Desiderio Roman, Thomas J. Ryan, William Sheehan, Calvin Smyth, John Speese, Margaret Sturgis, William B. Swartley, Roscoe Teahan, T. Turner Thomas, Martha Tracy, Stephen E. Tracy, Frank White.

An interesting program of papers, round table conferences, and practical demonstrations dealing with the problems related to the hospital standardization program of the College and hospital efficiency in general is being prepared for the annual hospital conference which opens at 10 o'clock on Monday morning in the grand ballroom of the Bellevue-Stratford Hotel. The conference will continue Monday afternoon and on Tuesday and Wednesday morning and afternoon. The program is planned to interest surgeons, hospital trustees, executives and nurses, and an invitation to attend is extended to all persons interested in the hospital field.

General headquarters for the Clinical Congress will be established at the Bellevue-Stratford Hotel located at the corner of Broad and Walnut Streets. All of the rooms on the second floor, including the grand ballroom which will be used for the evening scientific meetings, hospital conference on Monday, the annual meeting and other large gatherings, together with additional rooms on the roof, have been reserved for the use of the Congress and will be utilized for scientific meetings, conferences, film exhibitions, registration and ticket bureaus, bulletin boards, executive offices, scientific and technical exhibitions, etc.

An application for reduced railroad fares on account of the Congress in Philadelphia is pending before the railway passenger associations and it seems assured that a rate of one and one-half the regular first-class one-way fare will be in effect from all points in the United States and Canada.

PHILADELPHIA HOTELS AND THEIR RATES

The following hotels are recommended by the Committee on Arrangements:

	Minimum Rates with Bath	
	Single Room	Double Room
Adelphia, 13th and Chestnut Sts	$ 4.00	$ 7.00
Barclay, Rittenhouse Square East	6.00	8.00
Bartram, 33rd and Chestnut Sts		8.00
Belgravia, 1811 Chestnut St	4.00	7.00
Bellevue-Stratford, Broad and Walnut	5.00	7.00
Benjamin Franklin, 9th and Chestnut	4.00	7.00
Colonial, 11th and Spruce Sts	4.00	7.00
Drake, 1512 Spruce St	5.00	8.00
Elks, Broad and Vine Sts	2.50	5.00
Gladstone, 11th and Pine Sts	4.00	7.00
Green's, 8th and Chestnut Sts	3.00	4.50
Lorraine, Broad and Fairmount Ave	5.00	9.00
Maidstone, 1327 Spruce St	3.00	5.00
Majestic, Broad and Girard Ave	4.00	6.00
Mayfair, Lincoln Drive and Johnson St.	5.00	8.00
Pennsylvania, 39th and Chestnut Sts	3.00	5.00
Rittenhouse, 22nd and Chestnut Sts	3.00	5.00
Ritz-Carlton, Broad and Walnut Sts	6.00	10.00
Robert Morris, 17th and Arch Sts	3.50	5.00
St. James, 13th and Walnut Sts	3.50	5.00
Stephen Girard, 2027 Chestnut St	3.00	5.00
Sylvania, Juniper and Locust Sts	4.00	8.00
Tracy, 36th above Chestnut St	3.00	5.00
Walton, Broad and Locust Sts	3.50	5.00
Warwick, 17th and Locust Sts	5.00	8.00
Wellington, 19th and Walnut Sts	5.00	6.00
Westbury, 15th and Spruce Sts	5.00	10.00

LIMITED ATTENDANCE—ADVANCE REGISTRATION

Attendance at the Philadelphia session will be limited to a number that can be comfortably accommodated at the clinics, the limit of attendance being based upon the result of a survey of the amphitheaters, operating rooms, and laboratories in the hospitals and medical schools to determine their capacity for accommodating visitors. Under this plan it will be necessary for those who wish to attend to register in advance.

Attendance at all clinics and demonstrations will be controlled by means of special clinic tickets. This plan provides an efficient means for the distribution of the visiting surgeons among the several clinics, and insures against overcrowding, as the number of tickets issued for any clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.